THE UNITED STATES PATENT AND TRADEMARK OFFICE

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Serial No: **09/894,174** Group Art: 3626

Date Filed: June 27, 2001 Examiner: R. Rines

For: **MANAGEMENT METHOD OF PERSONS AT RISK OF COMPLICATIONS OF ARTERIAL OCCLUSIVE DISEASE**

Commissioner of Patents
P.O. Box 1450
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DECLARATION

William L. Blackshear, Jr., being duly sworn deposes and says that he is one of the inventors who, on June 27, 2001 filed the above identified application; that they completed their invention and disclosed the same to others in this country prior to September 1, 2000, as evidenced by the Letter Of Agreement between Tri-Med Management, Inc. and HealthHelp, Inc., dated July 8, 1999 (copy attached), more than one (1) year before the filing date of the application from which Crutchfield Patent No. 6,699,193 matured; that he does not know and does not believe that the invention had been in public use or on sale in this country, or patented or described in a printed publication in this or any foreign country for more than one year prior to their application, and they have never abandoned their invention.

Specifically, the invention referred to in the July 8, 1999 letter related to a classification and management system for patients with lower extremity arterial

occlusive disease. The system that comprises the following steps already developed at that time.

Specifically, the system comprises examining a patient at a healthcare facility with lower extremity arterial occlusion disease,

- collecting patient data including patient diagnoses, pertinent physical findings and noninvasive arterial pressure and blood flow data,
- recording the collected patient data,
- transmitting said collected patient data to an evaluating authority,
- comparing said collected patient data against a medically accepted set of disease specific criteria at the evaluating authority to provide an initial diagnosis and preliminary classification of those patients "potentially at risk" and those patients "not at risk" of developing complications of arterial occlusive disease,
- transmitting said preliminary classification to the healthcare facility,
- referring those patients classified as "potentially at risk" of arterial occlusive disease to an accredited laboratory for noninvasive vascular evaluation,
- evaluating those "potentially at risk" patients at the accredited laboratory against medically accepted criteria,
- recording the results of said noninvasive vascular evaluation at the accredited laboratory,
- transmitting said recorded results to the evaluating authority for final classification,
- classifying each patient at the evaluating authority against medically accepted criteria as "at risk" or "not at risk",
- transmitting said "at risk" or "not at risk" patient final classification to the healthcare facility,
- recording said "at risk" or "not at risk" patient final classification at the healthcare facility,
- referring patients having a final classification of "at risk" for critical ischemia with associated extremity lesions and patients with noninvasive evidence of severe ischemia to a vascular surgery facility for vascular surgical assessment to determine whether revascularization is necessary,
- assessing such "at risk" patients against medically accepted criteria as "clinical indication for operation" or "no indication for operation" at the vascular surgery facility,

- transmitting patient assessments assessed as "clinical indication for operation" or "no indication for operation" assessment to the evaluating authority,
- informing those patients assessed as "clinical indication for operation",
- electing either revascularization and periodic management system evaluation at the healthcare facility or routine wound care and periodic revaluation at the healthcare facility by patients assessed as "clinical indication for operation",
- monitoring patients assessed as "no indication for operation" by the healthcare facility with increased precautions to monitor for detection of any deterioration that would require reassessment,
- referring patients having ulcers, pain or gangrene at the time of "no indication for operation" assessment for reassessment,
- recording the reasons for not referring such patients as "clinical indication for operation",
- referring patients classified as "no indication for operation" that develop ulcers, pair and/or gangrene to the vascular surgery facility for reassessment,
- reassessing the referred patient at the vascular surgery facility against medically accepted criteria as "no indication for operation" or "clinical indication for operation",
- transmitting the reassessment of "no indication for operation" or "clinical indication for operation" to the evaluating authority for reevaluation as "no indication for operation" or "clinical indication for operation",
- transmitting the reevaluation to the healthcare faculty with the appropriate medical procedure and regimen,
- treating and monitoring patients classified as "not at risk", "at risk" and assessed as "no indication for operation" or "clinical indication for operation" at the healthcare facility,
- providing "not at risk" patients without limb ulcers routine care and precautions at the healthcare facility,
- providing "not at risk" patients with limb ulcers routine wound care at the healthcare facility,
- providing "not at risk" patients with limb ulcers periodic reevaluation by the evaluating authority,
- providing "at risk" patients assessed as "no indication for operation" or "operation not elected by patient", and "clinical indication for operation" patient undergoing revascularization at the vascular surgery facility with intensive wound care at the healthcare facility, and,
- providing periodic reevaluations of "at risk" patients assessed as "no indication for operation" or "operation not elected by patient" with increased precautions at the healthcare facility.

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true and further that these statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under 18 U.S.C. 1001 and that such willful false statements may jeopardize the validity of the application or any patent issued thereon.

William M. Blackshear, Jr.

William M. Blackshear, Jr.

LETTER OF AGREEMENT

After numerous conversations with Dr. Blackshear and Dr. Hagemeier there appears to be opportunities for HealthHelp, Inc., (HHI) and Tri-Med Management, Inc., (TMM) to work together. One area of interest for both parties is Beverly Nursing Homes, (BNH). In the event Beverly desires a comprehensive radiology vendor to service their residents in a fee for service or captivated arrangement, HHI would be interested in working with TMM to provide such services to BNH. Until we learn more about the potential client's desire to work with a radiology vendor the specifics of the partnership cannot be addressed. This letter serves as acknowledgment of the arrangement and involvement of HHI and TMM with regards to Beverly. It needs to be approved by TMM and will be an adjunct to their business not a competitor. TMM and their agents will not use any HHI proprietary information or products without written consent from HHI and will treat the information as confidential and will not try to compete with HHI or work with an HHI competitor with the same client. Likewise, HHI and their agents will not use any TMM propriety information or products without written consent from TMM and will treat the information as confidential and will not try to compete with TMM or work with a TMM competitor with the same client.

Both organizations require signing their Confidentiality and Technology Rights Agreement, which will be completed after review by their respective attorneys and before proceeding beyond the initial meeting with Beverly Nursing Homes.

By: William M. Blackshear

Dr. William M. Blackshear, Jr., M.D.

President

Tri-Med Management, Inc

Dated this 8th day of July, 1999

By: C Lee for RLS

CHARLES DUDLEY LEE

Robin L. Smith, MD, MBA

Chief Medical Officer

HealthHelp, Inc.

Dated this 8 day of July, 1999

Chapter 8

NONINVASIVE STUDIES OF PERIPHERAL VASCULAR DISEASE

JAMES S. T. YAO

Plethysmography

Plethysmography probably is the oldest method for measuring blood flow. In this method, measurements are made of changes in volume of an organ or region of tissue. The word "plethysmography" was derived from the Greek and literally means "the recording of increase." Swammerdam, in 1737, is credited with being the first to introduce this principle. It was not until the introduction of venous occlusion plethysmography, however, that measurements were made on a qualitative basis dependent on the alteration of pulse waveforms.

In the modern practice of vascular surgery, the use of plethysmography has been expanded to include detection of not only arterial occlusive disease but also carotid artery disease and venous problems. The use of oculoplethysmography and impedance plethysmography will be discussed in Chapters 10 and 11, respectively, and hence will not be discussed here. In addition to clinical application, plethysmography has been shown to provide valuable information on both arterial and venous physiology.

Several types of plethysmographs are now available for clinical use in the evaluation of arterial occlusions. These are volume, strain-gauge, and photoelectric plethysmographs. The water-filled volume recorder, popular in the early use of plethysmography, is now obsolete and has been replaced by the air-filled volume plethysmograph, notably, the pulse-volume recorder developed by Raines (1). For clinical application. the newer plethysmographs, such as the strain-gauge, photoplethysmograph, and pulse-volume recorder, are now standard equipment in many vascular laboratories.

INSTRUMENTATION

Strain-Gauge Plethysmograph

Strain-gauge plethysmographs use fine-bore silicone rubber tubes filled with mercury or a liquid-metal alloy that makes contact with copper electrodes at either end. The gauge is wrapped around the part being studied. As the part (calf or forearm) expands or contracts, the length of the gauge increases or decreases by a corresponding amount. The changes in length of the gauge reflect changes in the circumference of the part and are interpreted as volume changes. Because the resistance of the liquid-metal column varies with its length, changes in voltage across the gauge, when amplified, can be calibrated to reflect changes in circumference (volume) of the circumscribed part.

Changes in strain-gauge length can be calibrated mechanically to reflect volume flow, or electrical calibration by a bridge circuit can be used to measure voltage drop across the gauge.

Photoelectric Plethysmograph

The term *photoelectric plethysmograph* was first introduced by Hertzmann (2), who used it to measure blood volume changes induced in the skin by exercise in normal individuals and in patients with peripheral arterial disease. Early instrumentation included the use of a photo cell for pulse recording by means of alternating current (ac) signals. Recently, an infrared sensor has been used in the newer generation of photoelectric plethysmographs. In this newer instrument, a direct current (dc) coupling technique is also provided. The dc signal is not related to the pulse, is slow to change, and depends on changes in the total blood volume in the skin. With refinement in probe design, the photoplethysmograph has become a standard instrument for the study of venous problems and for the recording of pulse waveforms in most laboratories. The use of the dc mode to detect volume changes is helpful in recording finger or toe pressure.

Air-Filled Plethysmograph

One of the popular instruments used in the noninvasive laboratory is the pulse-volume recorder. In essence, this is an air-filled, segmental plethysmograph designed to measure segments of the leg. The air-filled plethysmograph uses a pneumatic cuff placed around the part being examined, often at several levels. By injecting a known volume of air into the cuff bladder, instantaneous volume changes in the limb segment beneath the cuff can be calibrated and recorded with a sensitive pressure transducer to provide pulsatile flow tracings. Changes in the configuration of such pulse-volume tracings indicate proximal arterial occlusion. By visual inspection, the pulse waveforms can be related to different degrees of ischemia (Figure 8-1).

In addition to recording arterial pulse-volume waveforms, the pulse-volume recorder can be converted for recording venous outflow and capacitance using the dc mode. Also, the instrument is useful to record penile pulse waveforms and to record ophthalmic

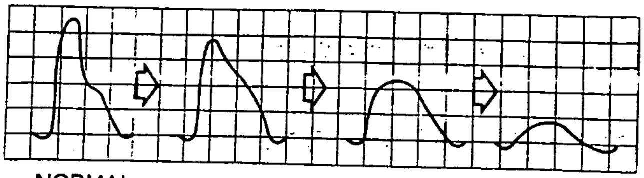

FIGURE 8-1. Changes in waveforms recorded by the pulse-volume recorder with progressive arterial disease. Severe ischemia is manifested by marked damping of the waveform. [From Kempczinski RF: Segmental volume plethysmography: The pulse volume recorder. In Kempczinski RF, Yao JST (eds): *Practical Noninvasive Vascular Diagnosis*. Chicago, Year Book Medical Publishers, 1982, pp 105–117.]

artery pressure by methodology similar to the Gee-OPG technique. (See Chapter 10.)

CLINICAL APPLICATIONS

Clinical use of plethysmography includes (1) determination of segmental systolic arterial pressures in the upper or lower limb, including the digits, (2) determination of limb blood flow (volume) expressed in milliliters per minute per 100 g of tissue, (3) pulse registration of the fingers or toes, (4) penile waveform analysis, and (5) testing for the thoracic outlet syndrome.

Systolic Pressure Recording in Limbs

Although Doppler ultrasound methods have largely replaced the use of the plethysmograph for indirect measurement of limb or digital systolic arterial pressure, there are applications when recordings of toe pressure or finger pressure are required. The plethysmograph (strain-gauge or photoelectric) in a dc mode is of particular use to record finger or toe pressures because signals may be inaudible by Doppler in a low flow state. The dc mode is designed to detect instantaneous changes of volume by a shift of the baseline in the recorder. Such a shift is often used as the end point for systolic pressure determination during inflation of the pressure cuff. This technique is also useful to study finger pressure and its response to cold stimulation (3).

Limb Blood Flow

Recording of limb blood flow requires the use of the venous occlusion technique introduced by Brodie and Russell in 1903 (4). Since then, numerous studies have appeared on measurement of quantitative blood flow using this principle. In 1949, Whitney (5) first introduced the mercury-in-rubber strain-gauge for venous occlusive plethysmography, and this simple method has emerged as a standard plethysmographic technique for recording limb blood flow.

Limb volume changes instantaneously with the rate of arterial inflow and venous outflow. If a blood pressure cuff is placed around the thigh and then inflated to a suitable pressure, the venous outflow from the limb will be stopped without altering the rate of arterial inflow. The resulting increase in limb volume distal to the cuff is caused by a similar increase in blood volume. Initially, the rate of increase in venous blood volume is equal to the rate of arterial inflow. The limb volume change per minute is expressed as a percentage increase, or milliliters per 100 mL per minute.

In principle, the segment to be measured is encircled by a fine elastic tube filled with mercury, which is connected to an electrical resistance-measuring circuit. The measuring circuit detects change in the length of the gauge. According to the formula of Whitney (5), changes in the volume of the segment are derived from changes in its circumference. With no significant tissue compression beneath the gauge, the percentage change in gauge length per minute is equal to a similar change in the segment circumference. Assuming that the segment length (length of the limb) is constant, the percentage change in cross-sectional area per minute is equal to a similar percentage change in volume per minute (milliliters per 100 mL per minute). With the use of a Wheatstone bridge, the changes in strain-gauge length are represented as changes in electrical resistance that can, in turn, be graphically displayed on any standard recorder.

Calibration of the strain-gauge is done by a mechanical method (6). The gauge is calibrated by changing the length by a known amount with the adjustment screw and noting the resulting recorder deflection (X mm). The calibration deflection (X mm) is compared with the flow deflection (Y mm) to give actual changes in gauge length per minute following proximal venous occlusion. This is compared with the original gauge length and expressed as a percentage change in circumference per minute (equivalent to one-half the percentage change in segment volume per minute). Other investigators prefer electrical calibration, which allows "on limb" calibration to be carried out at any time during a period of flow measurement without handling the gauge.

Both calf and foot blood flow can be measured simultaneously by placing the gauge around the calf and the foot. Because of the makeup of tissue in the calf, composed largely of muscle, it is generally thought that calf blood flow is a representation of muscle blood flow. Similarly, the relative lack of muscle in the foot means that flow recorded there is representative of the skin blood flow.

Recording of the volume flow using milliliters per 100 g of tissue per minute obtained at the calf (muscle) or foot (skin) has long been of interest to physiologists studying vasomotor tone of the lower extremity in both health and disease states. For the study of the upper extremity, both forearm and digital flow may be recorded by a similar technique. In addition to resting flow, measurement of reactive hyperemic flow may be accomplished using a pressure cuff inflated above the systolic pressure for 3 to 5 minutes. Reactive hyperemic flow is designed primarily to evaluate the status of the vascular bed distal to the site of examination. Both resting and reactive hyperemic flows are of value in the study of vasomotor tone and the effect of vasodilating agents in the forearm or digits (fingers or toes). For occlusive arterial disease of the lower limb, however, resting flow has neither diagnostic nor prognostic value (7). Conversely, reactive hyperemic flow provides better information. Storen (8) as well as others (9,10) have shown the extent of hyperemic flow to be of diagnostic value in patients with occlusive arterial disease. Strandness and Bell (11) and Myers and Irvine (12) have shown a significant correlation between an increase (> 50 percent) in pulse amplitude or volume flow during hyperemia and beneficial effects of lumbar sympathectomy.

In the clinical practice of vascular surgery, the use of calf (muscle) or skin (foot) flow is of limited value. Flow measure-

ment may be useful in the evaluation of the acute effect of various pharmacologic agents.

Pulse Registration of Fingers or Toes

Strain-gauge, air-filled volume- and photoplethysmographs are useful to record the pulse volume of fingers or toes or a segment of the limb as an organ. Because the contour and volume of these pulses have diagnostic significance, this qualitative information has been used to supplement diagnostic information from indirect pressure determinations. Normal waveforms have a sharp upstroke and a distinct dicrotic notch (Figure 8-2). In the presence of occlusive arterial disease, pulse waveforms recorded distal to the occlusive process become rounded, with slow acceleration and loss of the dicrotic notch (Figure 8-2). In the fingers, a special configuration of a so-called peaked pulse (Figure 8-2*A*) may be seen in patients with small-vessel involvement, such as with collagen disease, Buerger's disease, frostbite, and traumatic arteritis (13). This peaked pulse has a fairly rapid upswing, an anacrotic notch or bend, and a dicrotic wave located high on the downslope. In addition to visual inspection of pulse contour, the ability of the peripheral arterioles to dilate can be assessed by a change of contour following reactive hyperemia. This is done by placing a pneumatic cuff around the finger proximal to the gauge and inflating the cuff to a pressure above the brachial systolic pressure. After a 5-minute period of ischemic arrest, the cuff is suddenly deflated, and the pulses are continuously recorded until maximal hyperemia is reached. In normal individuals, an increase of pulse contour (flow acceleration) is observed. In the presence of obstruction proximal to the digits, peak volume will be delayed.

Failure of the pulse volume to increase indicates the presence of fixed resistance either due to stiff arterioles or because maximal hyperemia has already occurred. In either case, vasodilatation or surgical sympathectomy would not be effective in these patients. In multilevel occlusive disease, the reactive hyperemic response may show a decrease of pulse volume, and this finding indicates the presence of severe ischemia.

Digital pulse recording may also be of use to assess sympathetic activity in patients with vasospastic disorders. This is done by observing the change of pulse contours during deep breathing, with ice placed on the forehead, or while the patient performs mental arithmetic. An increase of pulse volume suggests the presence of sympathetic activity. For quantitative analysis, pulse contour may be subjected to Fourier analysis (14).

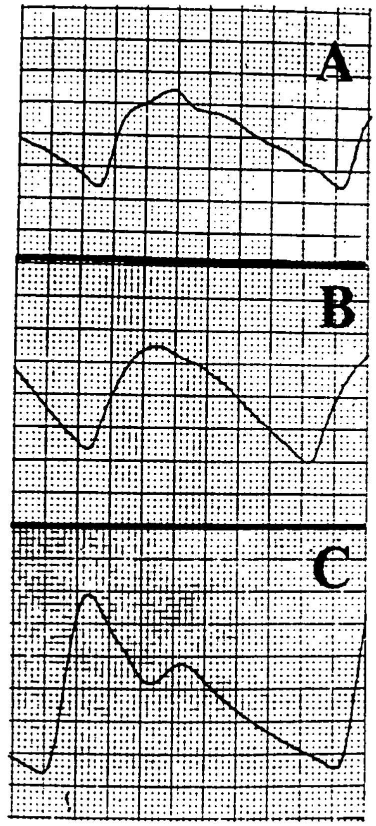

FIGURE 8-2. Finger pulse contour. *(A)* Peaked. *(B)* obstructive, *(C)* normal. (From Summer DS, Strandness DE: An abnormal finger pulse associated with cold sensitivity. Ann Surg 1972. 175:294–298.)

Penile Waveform Analysis

The use of the pulse-volume recorder or impedance plethysmograph to register penile pulse waveforms has been reported to be of value in the diagnosis of vasculogenic impotence (15,16).

Thoracic Outlet Testing

The photoplethysmograph sensor is a convenient tool to evaluate arterial thoracic outlet compression. The sensor is placed on the digits, and a change of pulse contour during various thoracic outlet maneuvers indicates the presence of thoracic outlet compression.

Doppler Segmental Pressure Determination in the Limbs

Since the introduction of the Doppler technique, recording of systolic pressure using a plethysmograph has received less attention. The Doppler technique is ideal for bedside, office, and intensive care monitoring. Although the technique of recording systolic pressure with the Doppler method is simple, the principles of measuring blood pressure must be observed in order to obtain accurate readings.

EQUIPMENT AND TECHNIQUE

Proper blood pressure cuffs, manometers, and control valves are prerequisites for accurate blood pressure recording. Regardless of the type of instrument used, the cuff size applied to the limb is of paramount importance in achieving accurate readings. An inflatable bladder is surrounded by an unyielding cover, called the cuff. The width of the bladder is critical. If it is too narrow (undercuffing), the blood pressure reading will be erroneously high, and if it is too wide (overcuffing), the reading may be too low. The American Heart Association (AHA) now recommends that the cuff size be based on limb circumference (17). It is recommended that the width of the inflatable bladder be 40 percent of the circumference of the midpoint of the limb, or 20 percent wider than the diameter. The length of the bladder should be twice its width (bladder length equal to 80 percent of the arm

circumference). Table 8-1 lists the recommendations for blood pressure cuff bladder dimensions made by the AHA in 1980 (17).

Since the recording of ankle pressure closely parallels the recording of brachial pressure, the bladder dimension and ankle circumference must be taken into consideration for accurate measurement. For measurement of ankle blood pressure, the conventional arm cuff is sufficient. Standard adult arm cuffs used in clinical medicine at the present time contain an air-inflatable rubber bladder that is 23 cm long and 12 to 12.5 cm wide, which should be sufficient for use at the ankle level. Measurements using cuffs of this size yield values that agree with intraarterial measurements.

The size of the thigh cuff is important. It should be 18 to 20 cm wide. For recording pressure of the lower thigh, we have found that a 19-cm cuff is useful. Barnes (18) has suggested a narrow cuff (11 cm) for upper thigh pressure measurement. In obese patients, a wider cuff may be more appropriate because of the large size of the thigh.

Digital cuffs for recording finger or toe pressures may be constructed of a bladder made of Penrose drain on a backing of nylon Velcro strip. Digital cuff width varies from finger to toe. The proper width for a finger is 2 to 2.5 cm and for the toe, 2.5 to 3 cm. Table 8-2 summarizes the different sizes of cuffs that should be used for recording lower limb pressure.

Two types of pressure registering systems are in general use: the mercury gravity and the aneroid manometer. Both give accurate and reproducible results when working properly. The aneroid manometer is probably more versatile for bedside or laboratory use. If an aneroid manometer is used, the instrument should be calibrated yearly by interposing a Y-connector in the tube from the cuff to a mercury manometer and attaching the sphygmomanometer to be tested to the free end of the connector.

All measurements are made with the patient in the supine position after a 10- to 15-minute period of rest. Brachial pressure is recorded with the Doppler method or with a conventional stethoscope before lower limb pressure measurement.

Ankle Systolic Pressure

The arm pressure cuff is applied snugly above the malleolus. The cuff is then inflated above the brachial systolic pressure (about 20 to 30 mmHg). The end point of systolic pressure is determined by the reappearance of the pulse (an audible sound by Doppler method or pulse waveform by plethysmography). Two or three measurements should be made on each limb.

TABLE 8-1. Recommended Bladder Dimensions for Blood Pressure Cuffs

Arm Circumference at Midpoint,* cm	Cuff Name	Bladder Width, cm	Bladder Length, cm
5–7.5	Newborn	3	5
7.5–13	Infant	5	8
13–20	Child	8	13
17–26	Small adult	11	17
24–32	Adult	13	24
32–42	Large adult	17	32
42–50†	Thigh	20	42

*Midpoint of arm is defined as half the distance from the acromion to the olecranon.

†In persons with very large limbs, the indirect blood pressure should be measured in the leg or forearm.

SOURCE: From Kirkendall et al. (17). Used by permission of the American Heart Association, Inc.

TABLE 8-2. Recommended Cuff Size for Lower Limb Pressure Measurement

Location	Cuff Width, cm
Adult upper thigh	11
Adult lower thigh	19
Adult thigh (contour-type cuff)	22
Adult ankle	12
Adult finger	2–2.5
Adult toe	2.5–3

SOURCE: Reproduced by permission from Yao JST: Noninvasive techniques of measuring lower limb arterial pressures. In Bernstein EF (ed): *Noninvasive Diagnostic Techniques in Vascular Disease*, 3d ed, St Louis, Mosby, 1985, pp 1–24.

Upper Thigh Pressure

A narrow cuff (11 cm) is placed just below the inguinal ligament, and the Doppler probe is placed over the popliteal artery for end point determination.

Lower Thigh Pressure

The thigh cuff is applied just above the knee. If the Doppler technique is used, the sound probe is placed over the popliteal artery to detect sound signals. It has been shown that placing the sensor close to the pressure cuff is critical to obtain accurate thigh pressure measurement, especially in patients with multiple-level occlusions (19). If plethysmography is used, pulse registration is normally done on the big toe. The end point of the systolic pressure is determined by the same maneuver used to obtain the ankle pressure.

Toe Pressure

A strain-gauge or photosensor is placed on the big toe to record pulse or volume changes (dc mode), and the cuff is placed at the base of the toe.

Postexercise Measurements

Ankle pressure can be measured after exercise in a similar manner. Standard treadmill walking is used. Immediately after termination of the treadmill exercise, the patient resumes the supine position, and ankle pressure is recorded at 1-minute intervals until the pressure reaches the preexercise level. Exercise testing is not advisable if the patient has angina or a severe arrhythmia. When indicated, monitoring of cardiac rhythm during treadmill exercise is needed to ensure safety of the procedure.

Postischemic Measurements (Reactive Hyperemia)

To simulate the reactive hyperemia induced by exercise, a pressure cuff applied to the thigh may be used to induce distal ischemia. The cuff is inflated to a level of 50 mmHg above the systolic pressure for a period of 3 to 5 minutes and then abruptly

deflated. Reactive hyperemia will follow immediately. Ankle systolic pressure is then recorded at 1-minute intervals until it returns to the resting pressure level.

CLINICAL APPLICATIONS

Segmental pressure measurements in the lower extremity include those at the high and low thigh, calf, and ankle levels. Also, toe pressure recorded by either the Doppler or photoplethysmograph may offer further hemodynamic information. Table 8-3 shows the normal values of systolic pressures commonly used in many vascular laboratories. Similar to all physiological measurements, ankle pressure is subject to variability, and this variation must be considered when evaluating the results of a longitudinal study. In our laboratory (20) as well as others (21), a change in the ankle pressure index of 0.15 is considered to be significant, but any change less than 0.15 is considered a result of measurement variation. Each laboratory must establish its own variability in this regard.

Diagnostic Use

The absence of a palpable pedal pulse often leads to the diagnosis of occlusive arterial disease. In borderline situations, such as in the presence of ankle edema or obesity, which make palpation of the pedal pulse difficult, the level of ankle pressure recorded by the Doppler technique often refutes or confirms the diagnosis instantly. In patients with occlusive arterial disease in whom the pedal pulses are not palpable, the Doppler flow velocity detector is able to detect flow signals from either the posterior tibial or dorsalis pedis artery. These flow signals represent the supply by collateral flow pathways, which could be used for registration of the systolic pressure end point by the indirect cuff technique.

Pressures measured at the thigh, calf, and ankle may aid in establishing the site or sites of occlusion. In the presence of iliac artery or common femoral artery occlusion alone, pressure recorded at these sites gives similar information, i.e., there is no pressure gradient. In contrast, a pressure gradient is noted between thigh and ankle levels when there is occlusion affecting the femoropopliteal segment. In multiple occlusions affecting the aortoiliac and femoropopliteal segments, different pressure gradients in the thigh and ankle are seen, with the ankle pressure being much lower than in patients with single occlusions.

In addition to establishing the diagnosis of arterial occlusive disease, recording of the ankle systolic pressure may help in detection of the popliteal artery entrapment syndrome. A marked decrease in ankle systolic pressure when the leg is placed in passive dorsiflexion and hyperextension suggests the presence of an entrapment syndrome. Also, when a steal phenomenon with an arteriovenous fistula is suspected, measurement of the ankle systolic pressure and observation of its change after compression of the fistula may help to confirm the diagnosis. An increase in systolic pressure with the arteriovenous fistula (proximal to the site of measurement) closed indicates a significant hemodiversion causing distal ischemia. With increasing use of the in situ vein graft, this diagnostic maneuver may help to determine the significance of an abnormal arteriovenous communication.

TABLE 8-3. Normal Values for Doppler Arterial Examination

Ankle systolic pressure	>Brachial systolic pressure (< 40 mmHg = limb-threatening ischemia)
Ankle pressure index (ankle-brachial ratio)	> 1.0
Thigh systolic pressure:	
High (narrow cuff)	30–40 mmHg > brachial systolic pressure
Low (wide cuff)	20–30 mmHg > brachial systolic pressure
Thigh pressure index	> 1.1
Pressure gradients	< 30 mmHg between adjacent sites
Toe systolic pressure	= to or 70% of brachial systolic pressure (< 30 mmHg = severe ischemia)
Toe systolic pressure index	0.7 ± 0.19 (0.35 ± 0.15 = claudication) (0.11 ± 0.10 = rest pain)
Penile pressure index	> 0.75 (0.60 = vascular impotence)
Finger systolic pressure index	≥ 0.95
Treadmill exercise test (2 mph, 12% grade)	Elevated or no decrease of ankle pressure after 5 min walking time

SOURCE: Modified from Pearce WH, Yao JST, Bergan JJ: Noninvasive vascular diagnostic testing. Curr Probl Surg 1983, 20(no 8). Used by permission of Year Book Medical Publishers, Inc., Chicago, pp 460–538.

Degree of Ischemia

Ankle systolic pressure, when compared with the brachial systolic pressure, gives an index of ischemia. Such a pressure index, as suggested by Winsor (22), has been shown to correlate significantly with initial clinical symptoms and with angiographic findings (23). The majority of patients with claudication have a pressure index of 0.59 ± 0.15. A low pressure index (0.26 ± 0.13) is often seen in patients with rest pain. In patients with impending gangrene, the pressure index is often extremely low (0.05 ± 0.08) or unrecordable (23). In multiple-level disease, the ankle systolic pressure is usually much lower than with a single-segment occlusion.

The absolute level of ankle systolic pressure and the pressure index are useful to express hemodynamic data. The pressure index helps distinguish normal from abnormal limbs, whereas the absolute pressure level helps in determining limb viability. Figure 8-3 illustrates the receiver operating characteristic (ROC) curves reported by Ouriel and Zarins (24) for these two parameters in the diagnosis of arterial occlusive disease. Ankle-brachial pressure index was a better predictor of pressure or absence of disease than absolute ankle pressure. Ankle systolic pressure, however, is more useful to determine limb viability. Ankle systolic pressure less than 40 mmHg is indicative of limb-threatening ischemia, and such a level has been recommended as an objective hemodynamic criterion for defining critical ischemia (25). However, this parameter alone should not mandate interventional treatment since some patients can tolerate such pressures for protracted periods without difficulty.

In patients with symptoms of limb-threatening ischemia, recording of the toe pressure is necessary when the ankle pressure exceeds 40 mmHg. Carter and Lezack (26) have found that

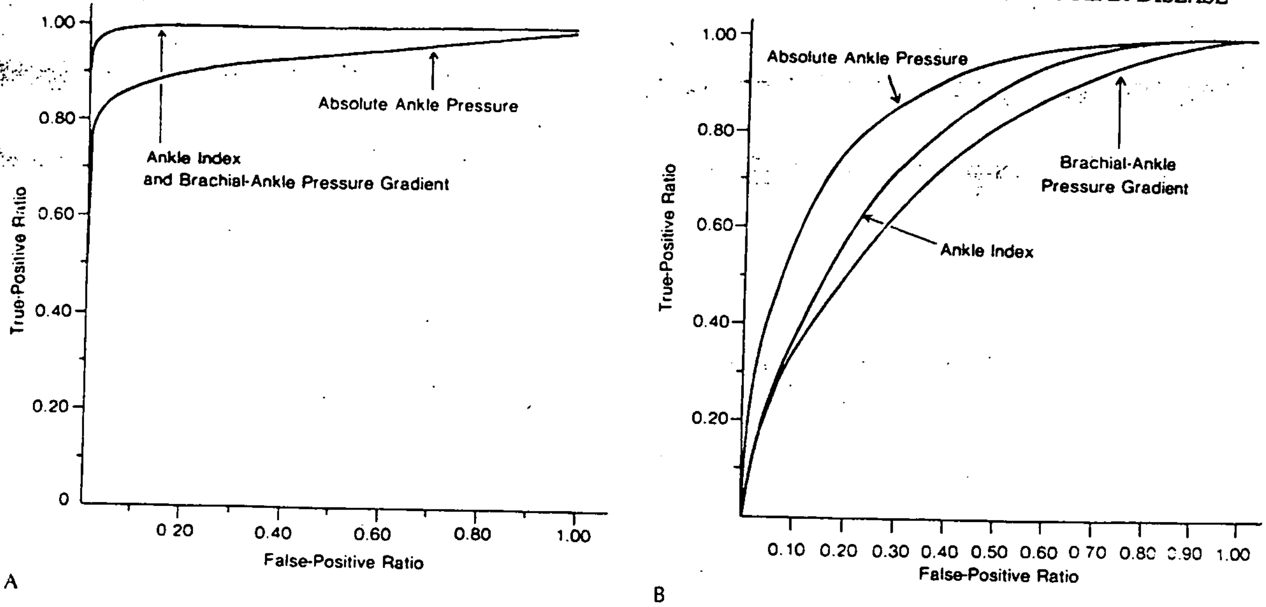

FIGURE 8-3. Receiver operator characteristic (ROC) curves illustrating the ability of absolute ankle pressure and ankle index to discriminate between normal and arteriographically diseased limbs *(A)*, and between viable and nonviable limbs *(B)*. (From Ouriel K, Zarins CK: Arch Surg 117:1297–1300, 1982. Copyright 1982, American Medical Association.)

systolic pressure in the toe was below 20 mmHg in 85 to 90 percent of limbs with rest pain, and below 40 mmHg in 100 percent. There was no difference between diabetics and nondiabetics. When arterial occlusion involves the plantar arteries, the toe pressure is a better index to establish the presence of severe ischemia. Also, toe pressure measurement is helpful in diabetic patients when an abnormally high ankle pressure is recorded. Toe pressure measurements may be subject to wide variation, however, and duplicate measurements may be needed for a reliable study (27).

The main objective in using the pressure index or critical level of ankle or toe systolic pressure is confirmation of the presence of severe ischemia. Although diagnosis of ischemic pain is not often difficult, the confirmatory finding of a low systolic pressure is rewarding, and it may help to differentiate ischemic pain from that of neurogenic origin, especially in patients with diabetic neuropathy. In high-risk patients, the decision to proceed with a limb salvage operation without objective documentation of severe ischemia may subject the patient to an unnecessary procedure.

Evaluation of Intermittent Claudication

Measurement of ankle pressure and observation of its change after standard treadmill exercise provide a simple test for the diagnosis of claudication of vascular origin. This is particularly true in patients initially having intermittent claudication who are found to have pedal pulses. These pulses, derived from collateral flow, often are associated with a decreased ankle pressure and often disappear after exercise. This "pulse-disappearing phenomenon" can be verified simply by recording the ankle pressure after exercise. When there is a significant drop in ankle pressure after exercise, the relationship between disability and arterial occlusion can be safely established. The relationship between postexercise ankle pressure and calf blood flow has been studied by plethysmographic and isotope techniques (28,29). A good correlation between flow and ankle pressure changes has been demonstrated. Since ankle pressure is easy to use, measurement of the ankle pressure response to exercise is now a standard laboratory test.

After treadmill exercise, the degree of ankle pressure drop and the time for the pressure to return to the preexercise level are the two important components that offer hemodynamic information. The degree of ankle pressure drop immediately after termination of exercise reflects the degree of inflow obstruction, whereas the recovery time denotes the adequacy of collateral pathways. In the presence of superficial femoral or femoropopliteal artery occlusion, the pressure drop is often to 50 percent of the preexercise level. In contrast, patients with iliac artery occlusion will have a greater drop in ankle pressure (70 to 90 percent) immediately after exercise because of shunting of blood to the thigh muscle group. The return of pressure to the preexercise level often occurs within 5 to 7 minutes because of the adequacy of collateral pathways in these patients. When two levels of occlusion are present, the picture becomes more complex. Following exercise, blood flow must traverse two collateral networks. A profound drop of ankle systolic pressure, often to an unrecordable level, results. Likewise, the recovery time is prolonged because of multiple levels of occlusion. An understanding of these hemodynamic changes helps determine the type of surgical corrective procedure to be used in these patients.

Treadmill exercise testing also helps to evaluate claudicants with an occult angiographic stenotic lesion or an inapparent stenosis on the anteroposterior (AP) view of the arteriogram. In

this circumstance, treadmill exercise establishes the hemodynamic significance of the iliac artery lesion.

Treadmill exercise is helpful in differentiating between atypical claudication and neurogenic claudication. A normal exercise response in a patient with exercise intolerance virtually eliminates a vascular etiology. In patients with coexisting neurospinal compression and arterial occlusive disease, a decision regarding appropriate therapy may be based upon the results of exercise testing (30).

Because of the nature of atherosclerotic disease, it is not surprising to find that patients with claudication may also have asymptomatic ischemic heart disease. The latter may assume clinical significance during stress testing. With the increasing use of the treadmill for assessing claudicants, the necessity for cardiac monitoring in these patients has become apparent. In a series of 100 consecutive claudicants who did not have any cardiac symptoms, the electrocardiogram was recorded at rest and during treadmill exercise. Evidence of ischemic heart disease was present in 56 percent of these patients at rest, and gross ischemic changes (for example, a 4-mm ST-segment depression), including arrhythmias, developed in 20 percent during exercise (31). A significant increase in cardiac arrhythmias in patients undergoing treadmill testing for claudication has been reported by others (32). In one study of 16 patients who developed abnormal ECG findings during exercise, 6 suffered a myocardial infarction after vascular reconstructive surgery (33). Although complications of the treadmill test are rare, the sequelae are potentially serious. To ensure safety and guard against litigation, treadmill exercise testing is best avoided in claudicants with overt cardiac abnormalities. Moreover, treadmill testing in claudicants should be done with cardiac monitoring and with resuscitation equipment and medical personnel trained in its use present in the laboratory.

The reproducibility of the standard treadmill test has been investigated in a series of 117 patients with stable claudication (34). Paired treadmill tests were done 1 week apart. The Doppler pressures were reproducible for the group as a whole, but there was a marked individual variation. The coefficient of variation was 15 percent for the pressures in the better leg and 24 percent in the more symptomatic leg. In the recent report by Ouriel and Zarins (24), a relative standard deviation of 15.6 percent for treadmill exercise and 14.7 percent for postocclusive reactive hyperemia was described. It was also found that brachial pressure after exercise was, on the average, 20 mmHg higher than the preexercise level (35). Failure of the brachial pressure to increase in response to exercise, or the finding of a decrease, is suggestive of poor myocardial performance.

Evaluation of Inflow (Aortoiliac Segment)

The importance of recognizing iliac artery stenosis before superficial femoral artery reconstruction has been stressed by many authors (see Chapter 28). Failure to correct a significant inflow lesion often results in occlusion of a femorodistal bypass or failure to improve hemodynamic status. When there is angiographic evidence of stenotic lesions affecting both the iliac and superficial femoral arteries, the crucial lesion producing the ischemic symptoms must be identified. Evaluation of inflow can be assisted by (1) common femoral artery waveform analysis, (2) thigh pressure measurement, (3) thigh-ankle pressure gradient, and (4) ankle pressure response to exercise. However, all these data must be correlated with clinical and arteriographic findings, and some groups find these noninvasive tests to be of little value in assessing combined segment disease (see Chapter 28). For claudicants, the use of treadmill testing offers more conclusive information.

To identify iliac artery stenosis, Bone et al. (36) have recommended the use of a narrow cuff to record upper thigh pressure. In our laboratory, as well as others (37), it has been found that the upper thigh pressure measurement adds little information on the status of inflow. The presence of a superficial femoral artery occlusion alters the result of upper thigh pressure recordings. Whereas a normal upper thigh pressure is generally reliable in ruling out hemodynamically significant aortiliac occlusive disease, an abnormal pressure does not differentiate between aortoiliac and superficial femoral artery disease. In the study by Flanigan et al. (37), it was found that the upper thigh pressure could not identify a significant iliac lesion when there was an associated femoropopliteal artery occlusion. Inconclusive thigh pressure readings may also be caused by artifacts of the thigh cuff (38) or the presence of a calcified superficial femoral artery. If there is difficulty in determining which lesion is hemodynamically significant, some authors have recommended intraoperative pressure measurement with pharmacologic vasodilatation (39,40).

Using the ankle pressure measurement and flow waveform recordings from the common femoral artery, the hemodynamic significance of anatomic disease in the iliac artery can be estimated. The presence of a monophasic common femoral artery waveform and the absence of a significant thigh-ankle pressure gradient indicate a significant inflow lesion. On the contrary, a segmental pressure measurement that shows a significant pressure gradient (greater than 30 mmHg) between thigh and ankle levels and a triphasic femoral waveform signify that the occlusion of the femoropopliteal segment is of hemodynamic significance. The degree of ankle pressure drop following treadmill exercise is also helpful in the evaluation of proximal arterial lesions. Since there is only minor shunting of blood to the thigh muscles, patients with femoropopliteal disease alone have only moderate reduction in postexercise ankle pressure. Conversely, inflow disease of the aortoiliac segment produces more profound drops in ankle pressure. When functionally significant disease is present in both arterial segments, ankle pressures often fall to zero following exercise and remain depressed for a prolonged period. Correction of the aortoiliac arterial occlusion often reverts the postexercise pattern to that of a single anatomic occlusion.

Obviously, the hemodynamic data must be correlated with presenting symptoms. For claudication, the use of exercise testing or reactive hyperemia is needed to achieve an adequate assessment. When a femorofemoral graft is considered, the adequacy of the donor artery inflow must be evaluated by treadmill exercise. A decrease in ankle pressure in the donor limb after exercise is ominous. In our analysis of 44 patients who underwent femorofemoral grafting for claudication, 20 (45 percent) of the patients had deterioration of donor limb hemodynamics after surgery. Seven of 14 patients who had abnormal treadmill exercise testing preoperatively had worsening of the ankle pressure response to exercise of the donor limb. On the other hand, a femorofemoral graft may remain functional and effective even

when there is less than perfect inflow, especially if the procedure is done for limb salvage. It would appear that strict patient selection criteria are needed when femorofemoral grafting is done for claudication.

Selection of Patients for Femorodistal Bypass

A low ankle pressure or unrecordable pressure does not indicate an unsalvageable situation. Several studies have now shown that the level of ankle pressure bears little relation to the prognosis of a femorodistal bypass (41,42). However, ankle pressure recording prior to reconstructive surgery below the inguinal ligament may be helpful (1) to supplement arteriographic findings and (2) to determine the exact site of distal anastomosis.

In the absence of acute occlusion, a low or unrecordable ankle pressure in patients with chronic ischemia caused by a femoropopliteal occlusion suggests the presence of severe occlusive disease distal to the popliteal trifurcation. Therefore, arteriographic examination must include the arteries in the calf and ankle region. In our recent analysis (43), the level of ankle pressure or the pressure index correlated well with the status of popliteal artery run-off in patients with normal aortoiliac inflow. In the presence of severe popliteal occlusive disease with involvement of the tibial or peroneal artery, the pressure index was often much lower (0.22 ± 0.13) than in patients with patent vessels distal to the popliteal trifurcation (0.55 ± 0.09). Also, the pressure gradient between the thigh and ankle helps to determine the severity of popliteal disease with tibial vessel involvement. When severe trifurcation disease is present, a large pressure gradient may develop between the lower thigh and ankle levels. In this situation, a reconstruction with the anastomosis placed into a tibial vessel to bypass the segments having the pressure gradient is desirable. A bypass graft to the popliteal artery proximal to diseased tibial and peroneal arteries is frequently associated with thrombosis (44), although this may not always be the case (see Chapter 26). In chronic ischemia, therefore, very low ankle pressure mandates complete arteriography including examination below the popliteal trifurcation.

The ankle pressure is helpful to supplement arteriographic findings when there is nonvisualization of arteries distal to the popliteal trifurcation on the preoperative arteriogram, especially the translumbar aortogram. The presence of a recordable pressure invariably indicates an operable situation (45), and nonvisualization by arteriography should not be an indication for amputation. Also, the difference between pressure recorded from the dorsalis pedis and posterior tibial arteries may help to determine the exact site of distal anastomosis. This is especially true in patients with inadequate visualization of distal arteries on preoperative arteriography.

Determination of Response to Sympathetic Ablation

The use of pressure studies to determine the response to sympathetic ablation will be discussed in Chapter 32, and hence, will not be repeated here. The importance of pressure measurements in relation to sympathectomy lies in the selection of patients who will certainly not respond to the procedure. Unless there is adequate distal perfusion (> 30 mmHg), lumbar sympathectomy is seldom effective. These patients, when identified preoperatively, will be spared an unnecessary operation (see Chapters 26 and 32 for even more restrictive views regarding sympathectomy).

Selection of Amputation Level and Prediction of Wound Healing after Minor Amputation

Determining the level of amputation for severe, uncorrectable ischemia remains a challenging problem, particularly after failed femoropopliteal or femorotibial grafting. A major objective of noninvasive technology has been to predict the most distal amputation that would heal primarily. Doppler segmental pressure measurement is a simple method to obtain helpful information. In a series of 66 patients who had major amputations, we used the ankle systolic pressure; the waveforms recorded from the posterior tibial, anterior tibial, and dorsalis pedis arteries; and the pressure in the lower thigh to determine the level of amputation (46). Our data suggest that, when there is no detectable flow in the popliteal artery, an above-knee amputation is advisable. On the other hand, detectable flow in the popliteal artery allows the recording of thigh pressure, and if the thigh pressure exceeds 50 mmHg, a below-knee amputation should be attempted because the chance for success is high. This has also been suggested by other authors (47,48). All patients who had audible signals at the posterior tibial or dorsalis pedis artery had a successful below-knee amputation. Of course, the use of pressure level alone may be fallacious, because the presence of infection and the operative technique strongly influence the surgical result. Nicholas (49), using a calf systolic pressure greater than 70 torr and an ankle pressure greater than 30 torr as an index that below-knee amputation would heal, found that this could not be achieved in 32 and 40 percent of cases, respectively.

Measurement of ankle pressure alone may not be sufficient to determine the success of minor amputation, including digit or forefoot amputation, and the use of toe pressure is needed to predict wound healing. Carter has stated that the chance of healing was nearly 100 percent in nondiabetic patients if digital pressure exceeded 30 mmHg (50). In diabetics, healing occurred in 75 percent of limbs with a pressure of 30 to 55 mmHg, and in 95 percent with higher pressures. A similar result has been reported by others, especially by Scandinavian investigators (51,52).

Photoplethysmography (PPG) may be used instead of Doppler ultrasound or strain-gauge recording. Using a PPG end point of 20 mmHg, Schwartz et al. (53) and Barnes et al. (54) observed uniform healing in all minor forefoot and digital amputations. Forefoot pulse-volume recording waveforms of only 4 mm or less have also been used to indicate that a toe or partial foot amputation will not heal (see Chapter 26).

Similar to selection of patients for lumbar sympathectomy, the purpose of pressure measurements to determine the level of amputation is to avoid an unnecessary amputation at a level where healing cannot occur.

Determination of Graft Patency and Graft Limb Occlusion after Reconstructive Surgery

The return of a pedal pulse usually indicates successful surgery, but the variability in palpation of pulses by different observers, especially in borderline cases and in the presence of postoper-

ative edema, often makes this method unreliable. Doppler ankle pressure measurement offers a simple and reliable method of assessing the patency of arterial reconstructions. Determination of ankle systolic pressure can be made as often as required and is of particular value in patients in whom a residual lesion is present, making the return of a palpable pedal pulse unlikely. This situation is often encountered in patients with occlusions affecting both the aortoiliac and superficial femoral arteries, where reconstruction is done only at the aortoiliac level. It may also be seen in patients with occlusion of the femoropopliteal artery and severe disease below the trifurcation of the popliteal artery. In deep femoral artery reconstruction, where there is a concomitant occlusion of the femoropopliteal segment, ankle systolic pressure recording is probably the only reliable, objective method to ascertain the result of operation.

Successful arterial reconstruction in single-segment disease (aortoiliac or superficial femoral) is always followed by an immediate increase in ankle pressure to a level close to a pressure index of 1.0 at the termination of the surgical procedure or within 6 hours postoperatively. On occasion, despite the absence of palpable pedal pulses immediately following the procedure, a delayed return of ankle pressure may be observed, especially with vein grafts. As long as the ankle pressure is not less than the preoperative level, continuous monitoring of pressure at hourly intervals will help to determine graft patency. Infrequently, the return of ankle pressure is delayed after aortoiliac reconstructions because of prolonged clamping of the aorta or excessive blood loss. In these cases, low thigh pressure recording is helpful to determine the success of the operation. Usually, after the effects of all anesthetic agents are dissipated 6 hours or more after surgery, ankle pressure returns to a higher level than before surgery. The use of thigh pressure is also helpful in composite-sequential bypasses, demonstrating the patency of the proximal bypass even when the ankle pressure is not improved after the procedure.

Ankle pressure recording is a simple method to monitor patency of arterial reconstructions. Perhaps the greatest value of this technique is in detection of failure when success has been assumed. Failure of a reconstruction is indicated by a steady drop in ankle pressure or ankle pressure index in the immediate postoperative period, or by failure of ankle pressure to improve to higher than the preoperative level in a 6- to 12-hour observation period. Early return to the operating room for correction of technical error often converts failure to success. The simplicity of ankle pressure measurement allows nursing personnel to perform hourly determinations in the recovery room and intensive care unit. Such measurements should be made routinely in addition to palpation of pedal pulses in all hospitals dealing with vascular reconstructive procedures.

Evaluation of New Treatment Modalities and Follow-up of Bypass Grafts

The use of noninvasive tests is essential in epidemiological settings for the diagnosis of atherosclerotic peripheral arterial disease and for recording the progress of the disease process. In an epidemiological study, Marinelli et al. (55) have found clinical examination of pulses to be totally unreliable. One-fifth of the patients with normal physical examination results were found to have abnormal results by ankle pressure determination. Recently, the American Heart Association Council on Epidemiology has recommended the use of Doppler ultrasound to record the ankle-arm pressure index before and after standard treadmill exercise as a diagnostic tool for field studies (56). The ankle pressure index can serve as baseline data to follow the natural history of the disease.

New therapeutic procedures are always being introduced as alternative treatments to surgery, and Doppler ultrasound examination provides an objective means to evaluate these procedures. The efficacy of transluminal balloon dilatation or thrombolytic therapy, for example, needs to be verified objectively. Several studies (57,58) have demonstrated that the results of balloon dilatation, when assessed by hemodynamic measurement, are inferior to those obtained by assessment of symptomatic improvement alone. The role of vasodilating agents in the treatment of claudication remains unclear. Evaluation of such vasodilator treatment can be obtained, since resting and exercise ankle systolic pressures provide objective tests to determine the efficacy of this pharmacologic approach (59). More importantly, assessment of the result of any type of reconstructive surgery by an objective technique is essential to establish the effectiveness of the procedure. For instance, we have found that the femorofemoral graft is effective in limb salvage situations, but that the procedure gives less than optimal results in patients with claudication (60). There has been concern about the effect of knee flexion on limb hemodynamics when prosthetic grafts are used, but ankle pressure measurement has demonstrated no ill effect during knee flexion (61).

In later follow-up, ankle pressure measurement has, in our hands, been the single most useful method to detect graft stenosis (62) and to confirm graft occlusion. A steady decrease in ankle pressure is indicative of progression of the disease distal to the bypass or of graft stenosis. The latter is frequently observed in patients with saphenous vein grafts.

A patent bypass graft by arteriography has been considered the hallmark of success of reconstructive arterial surgery. In contrast to this belief, a bypass graft may be patent by arteriography yet provide no functional improvement. This patent, but hemodynamically failed, graft can be detected by serial Doppler ultrasound examinations. A decrease in ankle pressure of 0.15 is considered to be hemodynamically significant and calls for repeat arteriography. A hemodynamically failed graft, if recognized early, may be corrected by surgery before graft thrombosis occurs (63). The frequent use of Doppler ankle pressure recording during the follow-up period should be routine in the assessment of the results of reconstructive arterial surgery.

Finally, postoperative or follow-up study by noninvasive techniques can determine whether an additional reconstructive procedure is necessary to achieve the desired result. The need for a further bypass procedure is often seen in patients who have multiple-level disease and in whom a proximal reconstruction is performed in the presence of a distal occlusion. If there is no improvement in the ankle pressure after aortoiliac or deep femoral artery reconstruction, a distal bypass graft to relieve symptoms is often warranted.

LIMITATIONS OF DOPPLER SEGMENTAL PRESSURE MEASUREMENT

In diabetics or in azotemic patients, it is not uncommon to find an abnormally elevated pressure because of calcification of the

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arterial wall. Systolic pressure may exceed 300 mmHg if there is an incompressible artery caused by circumferential calcification. Under this circumstance, recording pulse waveforms from the pedal arteries or toe pressure measurement will help to determine the status of ischemia. False-elevated systolic pressure is a limitation of indirect pressure measurement. On the other hand, the presence of a pressure > 300 mmHg invariably indicates arterial wall calcification.

Acknowledgments

This work was supported in part by the Conrad Jobst Foundation, the Seabury Foundation, and the Northwestern Vascular Foundation.

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5
Noninvasive Assessment of Peripheral Arterial Occlusive Disease

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As pointed out in the introduction to this section, peripheral arterial surgery should be designed to treat a physiologic rather than an anatomic defect. It makes little difference how aesthetically unappealing the arteriosclerotic plaque becomes if it does not restrict blood flow. The mere presence of disease, however widespread, rarely causes any problems. Aneurysms, with their potential for rupture, are, of course, an exception.

For these reasons, the surgeon who evaluates peripheral arterial disease must concentrate on the physiologic defects that the lesions produce. From the history, the surgeon gains an appreciation of how these defects limit the patient's activities. Unfortunately, the interpretation of symptoms is highly subjective, from both the patient's and the surgeon's point of view. Beyond a gross estimate of the physiologic limitations imposed by claudication or the suffering due to rest pain, the history yields nothing measurable. The physical examination affords more objectivity, in that pulses can be graded, ischemic ulcers measured, gangrenous areas delineated, and pallor, dependent rubor, and skin temperature noted. But even the most skilled surgical diagnostician will make some errors; and the ordinary physician will make many more.[10, 144, 160] Quite frequently, the physical examination fails to provide an accurate assessment of the severity of the disease.

Arteriography serves to define the anatomic lesion. Although good x-ray films are indispensable for most vascular surgery, they provide little objective data regarding physiologic disability. From clinical experience, the surgeon may have a fairly good idea of the type of limitations to be expected when the patient presents with an iliac occlusion, a superficial femoral occlusion, or multilevel disease. He also may be able to estimate the efficacy of the collateral circulation. Nevertheless, the experienced surgeon will recall many cases in which lesions shown on radiographs produced considerably more or less disability than expected.

Thus, a comprehensive assessment of atherosclerotic occlusive disease requires the integration of physiologic, anatomic, and clinical information. This chapter reviews the diagnostic information that can be derived from the noninvasive instruments and methods introduced in Chapter 4 and then discusses the application of this information to specific clinical situations.

Pressure Measurement

Measurement of pressure has distinct advantages over the measurement of flow for identifying the presence of arterial disease and for assessing its severity. Even though resting flow levels remain normal, there is almost always a pressure drop across an increased arterial resistance—even at rest.[40, 224, 251, 254] Pressure measurements can be made more sensitive by augmenting blood flow through a stenotic segment. This can be accomplished by exercise, by reactive hyperemia, or by the intra-arterial administration of vasodilating drugs. With increased blood flow, pressure drops are greater, and even those that were not noticeable under baseline conditions become evident (see Figs. 3–14 to 3–17).[13, 31, 41, 215, 254]

Ankle Pressure

Of all the noninvasive tests available for evaluating the functional severity of peripheral arterial disease, none is more useful than measurement of systolic blood pressure at the ankle. Not only does it provide a simple, reliable means of diagnosing obstructive arterial disease, but it is also readily applicable to follow-up studies.

The method has been described in Chapter 4. A pneumatic cuff is wrapped around the ankle and a Doppler probe is placed over the posterior tibial or dorsalis pedis artery. Ordinarily, the pressure measured at these two sites should differ by no more than 10 mmHg. A pressure difference greater than 15 mmHg suggests that there is a proximal occlusion or stenosis in the artery with the lower pressure.[40] The pressure at the site giving the highest value is taken as the ankle pressure.

At times, no audible Doppler signal can be obtained over either the posterior tibial or the dorsalis pedis artery. In these cases, a careful search will often reveal a peroneal collateral signal anteriorly, near the lateral malleolus. When no Doppler signal can be found, the ankle pressure can be measured with a plethysmograph placed around the foot or applied to one of the toes.

Normally, the systolic pressure at the ankle exceeds that in the arm by 12 ± 8 to 24 ± 9 mmHg.[26, 41, 140]

Table 5–1. Segmental Pressure Indices in Normal Subjects (mean ± SD)*

Author and Year	Thigh	Above Knee	Below Knee	Ankle
Carter (1968)[39]	—	1.16 ± 0.05§	—	1.15 ± 0.08‡
Yao et al. (1970, 1973)[254, 255]	—	—	—	1.11 ± 0.10
Wolf et al. (1972)[251]	—	—	—	1.09 ± 0.08†
Fronek et al. (1973)[77]	1.34 ± 0.27‖	1.32 ± 0.23‖	1.26 ± 0.24‖	1.08 ± 0.10‡
Cutajar et al. (1973)[58]	1.53 ± 0.17†	—	1.17 ± 0.13‡	1.08 ± 0.09‡
Hajjar and Sumner (1976)[91]	1.37 ± 0.20†	1.26 ± 0.11†	1.16 ± 0.10†	1.06 ± 0.08†
Rutherford et al. (1979)[193]	1.28 ± 0.17†	1.24 ± 0.17†	1.16 ± 0.17†	1.08 ± 0.17†

*Pressure index equals systolic pressure at site of measurement divided by brachial systolic pressure.
†Cuff 10 by 40 cm.
‡Cuff 12.5 by 30 cm (standard).
§Cuff 15 by 45 cm.
‖Cuff 17 by 50 cm.

This reflects the augmentation of the systolic pressure that occurs as the pressure wave travels peripherally. Distal to a hemodynamically significant lesion, the ankle pressure is almost invariably decreased.[26, 220, 224, 225] A single stenosis of 50 per cent or more or multiple mild irregularities of the arterial lumen will reduce the ankle pressure by at least 10 mmHg.[196] Typical ankle-arm pressure gradients are isolated superficial femoral obstruction, 53 ± 10 mmHg; isolated aortoiliac obstruction, 61 ± 15 mmHg; and multilevel obstruction, 91 ± 23 mmHg.[224]

Ankle Pressure Index

Because the ankle systolic blood pressure varies with the central aortic pressure, it is convenient to normalize the values by dividing the ankle pressure by the brachial blood pressure.[39, 40, 249, 258] This ratio, which is commonly referred to as the *ankle:brachial index* (ABI), normally averages about 1.10 when the well-rested subject is lying supine (Table 5–1). Although an occasional patient with functionally significant arterial stenosis will have an ABI that exceeds 1.00,[1, 39, 76, 171, 258] in the vast majority of patients with arterial disease, the resting index will be much lower.[1, 58, 251, 255, 258] In fact, an ABI less than 1.00 is highly suggestive of functional arterial obstruction;[39, 40, 58, 254, 255] only rarely does a normal limb have an index less than 0.92.[91, 171]

As shown in Figure 5–1 and Table 5–2, the ABI varies somewhat with the location of the arterial obstruction.[224, 251, 255] Values tend to be highest when the lesion is confined to the popliteal or below-knee arteries and lowest in limbs with multilevel disease.[40, 193, 251, 258] Carter found that the ABI exceeded 0.50 in 85 per cent of patients with a single block but was below 0.50 in 91 per cent of those with two or more blocks.[40] In addition, the ABI decreases as the functional severity of the disease increases, the lowest values being obtained in limbs with impending gangrene and the highest in limbs with mild claudication (Fig. 5–2).[58, 172, 180, 183, 254, 255] The ABI also correlates with arteriographic findings.[196, 258] Values are lowest when there is complete occlusion and highest when there is minimal atheromatous change (Fig. 5–3).[39, 58, 233, 258] As one would predict, based on the hemodynamic principles outlined in Chapter 3, the length of the occlusive process and the length of the bypassing collaterals are less important than their diameters.[63]

Since the ABI is reasonably stable from one examination to the next in the same individual (provided there is no change in the obstructive process), it constitutes an effective means of following the patient's course. A consistent decrease indicates advancing disease or a failure of arterial reconstruction.[22, 154, 155, 170, 228] A spontaneous rise in the ABI is usually attributable to the development of collateral circulation.[204, 205] After successful reconstructive surgery, there will be an increase in the ABI.[58, 217, 227, 228, 258] If all obstructions have been totally removed or bypassed, the index will exceed 1.0; however, if there are residual sites of obstruction, the ABI will increase although not to normal levels (Fig. 5–4).[227]

Technical Errors

Ankle pressure measurements are easily made and are remarkably free of error. The standard deviation between two measurements repeated within a few minutes is about 5 mmHg, and only 8 to 9 mmHg when the measurements are repeated from one day to the next.[163] These figures do not take into account varia-

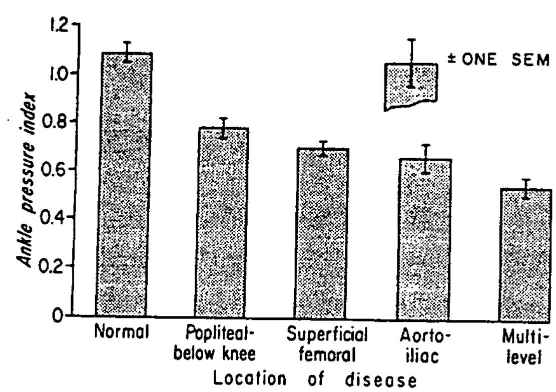


Figure 5–1. Resting ankle blood pressure indices (ankle systolic/arm systolic) measured in normal limbs and in limbs with arterial obstruction localized to different anatomic levels. (From Strandness, DE Jr, Sumner DS: Hemodynamics for Surgeons. New York, Grune & Stratton, 1975; data derived from Wolf et al, 1972.)

Table 5–2. Segmental Pressure Indices in Patients with Occlusive Arterial Disease of the Legs (mean ± SD)*

Location of Obstruction	Author and Year	Upper Thigh	Above Knee	Below Knee	Ankle
Aortoiliac	Fronek et al. (1973)[77]	0.72 ± 0.25	0.70 ± 0.24	0.62 ± 0.21	0.57 ± 0.18
	Rutherford et al. (1979)[193]	0.81 ± 0.25	0.76 ± 0.25	0.71 ± 0.25	0.68 ± 0.32
	Ramsey et al. (1979)[182]	0.81 ± 0.27	0.72 ± 0.25	0.59 ± 0.22	0.54 ± 0.22
Femoropopliteal	Fronek et al. (1973)[77]	1.26 ± 0.39	0.92 ± 0.39	0.73 ± 0.30	0.51 ± 0.28
	Rutherford et al. (1979)[193]	1.25 ± 0.27	0.86 ± 0.22	0.75 ± 0.18	0.65 ± 0.18
	Ramsey et al. (1979)[182]	1.19 ± 0.21	0.87 ± 0.23	0.70 ± 0.18	0.60 ± 0.19
Combined aortoiliac and femoropopliteal	Fronek et al. (1973)[77]	0.97 ± 0.34	0.79 ± 0.32	0.61 ± 0.28	0.48 ± 0.31
	Rutherford et al. (1979)[193]	0.89 ± 0.17	0.72 ± 0.17	0.58 ± 0.17	0.53 ± 0.28
	Ramsey et al. (1979)[182]	0.79 ± 0.21	0.62 ± 0.17	0.49 ± 0.15	0.39 ± 0.15

*Pressure index equals systolic pressure at site of measurement divided by brachial systolic pressure.

tions in central arterial pressure. When the ABI is considered rather than the absolute value, the day-to-day results are even more consistent. This test is also subject to interobserver and intraobserver variability, as well as to nonpathologic biologic variability. A change in the ABI of 0.15 or more almost certainly lies beyond the 95 per cent confidence limits of "normal" variation and therefore usually implies a significant physiologic change.[7, 112]

Medial calcification, which renders the underlying arteries incompressible, is responsible for most of the errors made in measuring ankle pressure.[76, 102, 180, 216, 229] Since patients with diabetes are particularly prone to this problem, one can anticipate that ankle pressure measurements in diabetics may be 5 or 10 per cent too high.[180] In these cases, it is sometimes possible to estimate the pressure by elevating the foot and noting the vertical distance from the bed at the point at which the Doppler signal disappears.[83] Multiplying this distance (in centimeters) by 0.735 will give the pressure in mmHg.

Confusion between arterial and venous flow can also occur when the arterial flow velocity is decreased and the signal becomes less pulsatile.[1] Venous signals can, however, be distinguished from arterial signals with the directional Doppler flowmeter. Moreover, venous signals can be augmented by foot compression (see Chapter 136), but arterial signals either are not affected or will diminish. If doubt still remains, a plethysmograph can be substituted to sense the return of flow as the cuff is deflated.

Segmental Pressure

Further diagnostic information can be obtained by measuring the pressure gradient down the leg.[100, 120, 216, 249] Only rarely do these measurements need to be made when the ankle pressure is normal.[76]

The following is but one of a number of techniques that have been advocated. Pneumatic cuffs (width 10

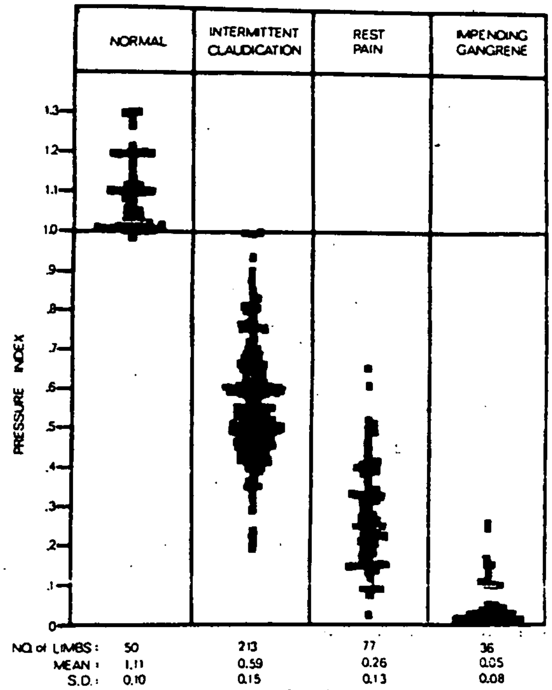


Figure 5–2. Relationship of ankle pressure index to functional impairment produced by the occlusive process. (From Yao ST: Br J Surg 57:761, 1970.)

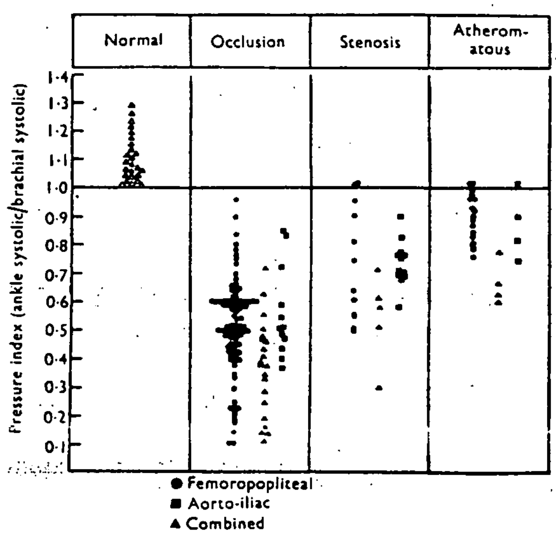


Figure 5–3. Relationship of ankle pressure index to the severity of the occlusive process. Note that the index exceeds 1.0 in all normal limbs in this series. (From Yao JST, Hobbs JT, Irvine WT: Br J Surg 56:676, 1969.)

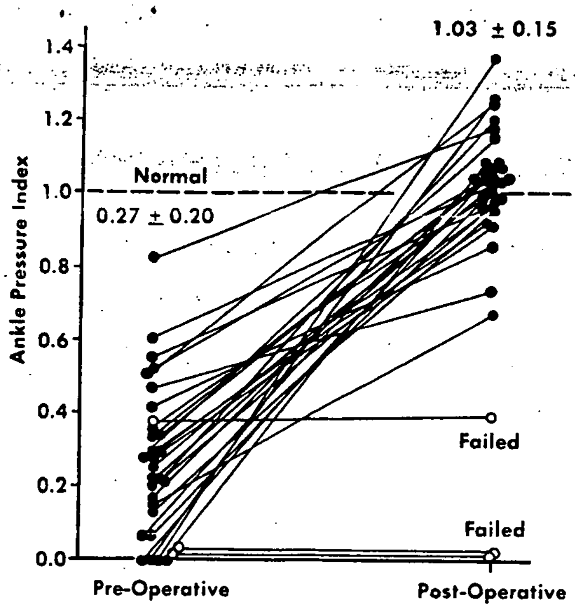


Figure 5–4. Results of femorotibial and femoroperoneal grafts. Ankle pressure indices before and after 31 bypass grafts from femoral to tibial, peroneal, or dorsalis pedis arteries. Open circles indicate grafts that failed within 30 days. Mean and standard deviations of the patent grafts are indicated. (From Sumner DS, Strandness DE Jr: Surgery 86:442, 1979.)

cm) are placed around the thigh at groin level, around the thigh just above the knee, around the calf below the knee, and at ankle level. Blood pressure is measured at each level by the method described in Chapter 4 (see Fig. 4–14).

Upper Thigh Pressure

In most normal individuals, blood pressures measured noninvasively at the upper thigh exceed those measured at the brachial level by 30 to 40 mmHg.[1, 58, 100, 249] Indices, obtained by dividing the thigh pressure by the brachial pressure, are comparably elevated, averaging around 1.30 to 1.50 (see Table 5–1).

It must be remembered that these values do not accurately reflect the femoral artery pressure, which, when measured by invasive techniques, is almost identical to the brachial pressure.[174] Moreover, as indicated by the standard deviations in Table 5–1, upper thigh pressures are highly variable even in normal subjects. Owing to the disparity between cuff width and thigh diameter, higher pressures are obtained in patients with large thighs and lower, more nearly accurate, pressures are obtained in patients with small thighs.[216]

A thigh pressure equal to or lower than the arm pressure usually indicates hemodynamically significant aortoiliac disease.[58] When the thigh pressure exceeds arm pressure but exceeds it by less than 15 to 30 mmHg, iliac disease may be suspected but could be absent if the diameter of the thigh is small.[100, 193] Comparison of the pressures obtained from the two thighs is of some value in these cases.[77] A 20 mmHg difference is said to be significant; however, the author and his colleagues have not found this difference to be a reliable indicator.[182]

Thigh pressure indices associated with aortoiliac obstructive disease are shown in Table 5–2 and Figure 5–5. It is apparent from Figure 5–5 that the thigh index may be lower than 1.0 in limbs with superficial femoral obstructions even when there is no hemodynamically significant disease in the aortoiliac segment.[25] This is somewhat more likely to occur in the presence of concomitant stenosis of the profunda femoris artery. Although the thigh index seldom exceeds 1.0 in limbs with total occlusion of the iliac artery, it is not uncommon to find normal indices in limbs with hemodynamically significant stenoses of the iliac arteries.[25] This is most likely to occur when the thighs are large. There is, however, another possible explanation. Since compression of the upper thigh by the cuff temporarily restricts arterial inflow, the pressure gradient across the external iliac artery is reduced. Consequently, when a stenosis is confined to this artery, the upper thigh reading may be spuriously high.[76]

If an upper thigh index of 1.0 is taken as the lower limit of normal, the data in Figure 5–5 indicate that there would be no mistakes in the normal control group; but in the patient groups, the sensitivity for detecting hemodynamically significant disease would be only 67 per cent, whereas the specificity for identifying the absence of disease would be 90 per cent.[193] Cutajar and coworkers reported that values over 1.20 are normal, those below 0.80 suggest complete occlusion, and those in between usually indicate the presence of aortoiliac occlusive disease.[58] According to these criteria, the data in Figure 5–5 show that only 10 per cent of the studies with indices exceeding 1.20 would be falsely classified as negative and that only 7 per cent of limbs with indices less than 0.80 would have no hemodynamically significant disease. Between these limits, however, significant disease was found in only 33 per cent. Moreover, 19 per cent of normal control limbs had indices below 1.20.

Pressure Gradients

Between any two levels in the normal leg, the pressure gradient usually does not exceed 20 to 30 mmHg (Table 5–3).[1, 216] Gradients greater than 30 mmHg strongly suggest that a significant degree of arterial obstruction is present in the intervening artery.[1, 81, 100] When the arterial segment is completely occluded, the gradient generally exceeds 40 mmHg.[77, 216] Rutherford and associates found that an upper thigh to above-knee gradient of 15 mmHg best distinguished limbs with superficial femoral occlusion from those without.[193] Similar gradients between the above-knee and below-knee levels and between the below-knee and ankle levels were found to have some predictive value related to disease in the popliteal and below-knee segments, respectively; but there was a great deal of overlap.

In addition to measuring "longitudinal" gradients along the leg, it is frequently helpful to compare pressures in one leg with those at the same level in the other leg. A "horizontal" difference of 20 mmHg in normotensive patients may be significant, implying greater disease at or above this level in the leg with the lower pressure.[77]

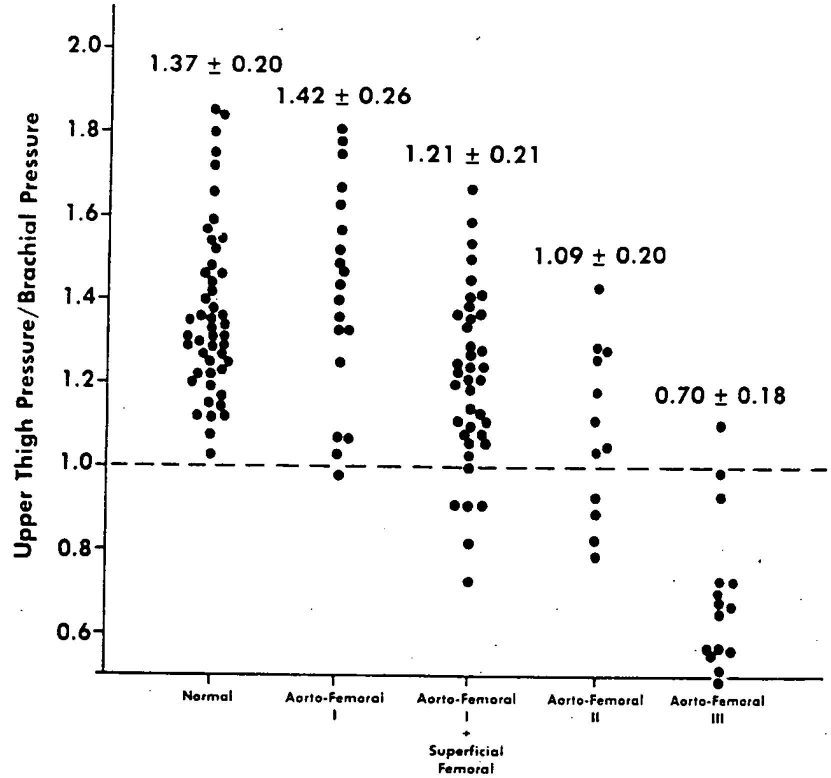


Figure 5–5. Identification of aortofemoral obstruction. Upper thigh index in normal limbs and in limbs with arteriosclerotic disease of the aortofemoral segment. Grading of aortofemoral disease is: I, less than 50 per cent diameter stenosis; II, more than 50 per cent diameter stenosis; and III, total occlusion. Grades II and III are hemodynamically significant.

The ratio of the pressure at all levels in the leg to that in the arm should exceed 1.0 (Tables 5–1 and 5–2). Values lower than this at any level imply significant obstructive disease in the proximal arteries. This is fairly reliable. Theoretically, by making both "longitudinal" and "horizontal" comparisons of the segmental pressures or indices, the examiner should be able to locate the site or sites of arterial obstruction and obtain some idea of their functional significance. Idealized values illustrating this point are shown in Table 5–4. Isolated disease in the aortoiliac or superficial femoral segments can usually be identified; but in limbs with multi-level disease, identification is frequently suboptimal. For example, superficial femoral obstructions may not produce an abnormal gradient in limbs with aortoiliac disease, iliac stenoses may not be recognized in limbs with superficial femoral disease, and below-knee disease is commonly misdiagnosed or overlooked when there is concomitant superficial femoral obstruction (Table 5–5).[100, 193]

Technical Errors

In an effort to achieve a more accurate assessment of the thigh pressure, some investigators have advocated using a single wide cuff (19 cm) rather than two 10 cm cuffs, one at the upper thigh and the other above the knee.[101] Gray and colleagues compared thigh pressures obtained with a wide cuff and direct measurements of the femoral arterial blood pressure in an effort to see how accurately the noninvasive pressure predicted aortoiliac disease.[88] A normal thigh-brachial index (exceeding 0.90) was generally reliable in ruling out inflow disease (only 13 per cent false-negative results). The thigh pressure, however, was distortedly low in 59 per cent of the studies, implying the presence of aortoiliac disease when in fact there was none. All of these false-positive errors occurred in limbs with occlusions of the superficial femoral artery. Thus, it would appear that the wide cuff is less accurate than the narrow cuff for diagnosing aortoiliac stenoses. Moreover, Heintz and

Table 5–3. Pressure Gradients in Normal Subjects (mean ± SD)

Author and Year	Arm–Upper Thigh	Upper Thigh–Above Knee	Above Knee–Below Knee	Below Knee–Ankle
Winsor (1950)[249]*	−22	13	11	8
Bell (1973)[18]†	−6 ± 12	2 ± 8	—	—
Hajjar and Sumner (1976)[91]‡	−46 ± 24	13 ± 19	12 ± 4	10 ± 9
Rutherford et al. (1979)[193]‡	−35 ± 18	5 ± 12	10 ± 15	11 ± 15

*Cuff 13 cm width.
†Cuff 18 by 60 cm.
‡Cuff 10 by 40 cm.

Table 5–4. Typical Segmental Systolic Arterial Pressures (mmHg)

	Normal	Arterial Disease: *Iliac*	*Superficial Femoral*	*Iliac and Superficial Femoral*	*Below Knee*
Arm	120	120	120	120	120
Upper thigh	160	110	160	110	160
Above knee	150	100	100	70	150
Below knee	140	90	90	60	140
Ankle	130	80	80	50	90

associates have shown that detection of superficial femoral disease by means of the "wide cuff to below-knee pressure gradient" is considerably less accurate than it is with the narrow cuff technique, which allows gradients across both the thigh and knee to be analyzed.[100] Others have reached similar conclusions.[72, 192]

Not infrequently, the pressure gradient between two adjacent segments of the leg may appear to be reversed. For example, the above-knee pressure may exceed that recorded at the upper thigh or the below-knee pressure may be greater than that recorded at the above-knee level. This reversal of the normal pattern of progression is usually due to local arterial incompressibility or to varying relationships between the size of the cuff and the limb.[1, 76] In hypertensive patients, the gradient between any two adjacent levels may be increased. On the other hand, when the cardiac output is low, the pressure drop may be diminished.[249]

Normal blood pressure gradients may be obtained in limbs with arterial obstructions when collateral channels are quite large. These findings do not really constitute errors, since the measurements are designed to reveal *functional* rather than *anatomic* obstruction.[216] For example, the pressure gradient from the below-knee level to the ankle is typically normal in limbs in which either the anterior tibial or posterior tibial artery is patent.[1, 104, 141, 202, 216]

Obstructions of arteries such as the internal iliac or the profunda femoris, which are not directly responsible for perfusion of the distal leg and ankle, may be missed.[1, 39] As pointed out earlier in this chapter, occlusions of the profunda femoris artery will become evident when the superficial femoral artery is also occluded. In these cases, the profunda femoris constitutes the major collateral channel supplying the lower leg and ankle. Therefore, if both of these arteries are obstructed, the upper thigh pressure will be abnormally low even though the aortoiliac segment is completely patent.

Because of the errors inherent in noninvasive assessment of the upper thigh pressure, direct femoral arterial pressure measurements are being used more frequently (see Chapter 6).[13, 31, 70, 152, 238] It should be noted, however, that direct pressure measurements are also subject to errors that are easily overlooked; most systems, for example, are underdamped, giving spuriously high systolic pressures.[35] Many laboratories also use segmental plethysmography or Doppler flow signal analysis to supplement segmental pressure studies.[180, 193]

Toe Pressure

Toe pressures are measured as described in Chapter 4.[52, 89, 137, 162] A pneumatic cuff of appropriate width (about 1.2 times the diameter of the digit) is wrapped around the proximal phalanx, and a flow sensor is applied distally. Although mercury strain-gauges work well for this purpose, photoplethysmographs, which are more stable and occupy less space on the tip of the digit, are generally more convenient to use.

At toe level, the systolic blood pressure is usually somewhat lower than the brachial pressure. According

Table 5–5. Accuracy of Segmental Pressures for Locating Arterial Obstructive Disease*

Arteriographic Diagnosis	Diagnosis Based on Segmental Pressure Data (Per Cent): *Normal*	*Aortoiliac*	*Aortoiliac and Superficial Femoral*	*Superficial Femoral*	*Superficial Femoral and Popliteal*	*Popliteal*
Normal	97.2	1.4	—	—	1.4	—
Disease						
Aortoiliac	12.5	75.0	12.5	—	—	—
Aortoiliac and superficial femoral	6.3	6.3	78.0	3.1	6.3	—
Superficial femoral	15.0	—	10.0	55.0	15.0	5.0
Superficial femoral and popliteal	8.0	—	4.0	24.0	60.0	4.0
Popliteal	57.0	—	7.0	—	—	36.0

Note: Popliteal includes popliteal artery or two or more of the peroneal-tibial arteries.
*Modified from Rutherford RB, Lowenstein DH, Klein MF: Am J Surg *138*:211, 1979.

to Nielsen and associates, in the supine position, toe pressures of young normal individuals averaged 4.8 ± 6.6 mmHg below those in the arm.[162] In older subjects, toe pressures were 9.8 ± 10.7 mmHg less than those in the arm.

Figure 5–6 shows the distribution of toe pressures in 296 limbs with arteriosclerosis obliterans.[183] No asymptomatic patient had a toe pressure less than 50 mmHg, and only 11 per cent of those whose complaints were limited to claudication had toe pressures less than 30 mmHg. In contrast, 81 per cent of the limbs with ischemic rest pain had toe pressures less than 30 mmHg, and none had pressures above 40 mmHg. Eighty-one per cent of the limbs with toe pressures less than 30 mmHg and almost all of those with pressures less than 15 mmHg had ischemic symptoms at rest. Patients with rest pain usually have toe pressures below 20 to 30 mmHg, but those with ischemic ulcers often have somewhat higher pressures.[43, 106, 183, 236]

Toe indices (toe pressure divided by brachial pressure) of patients with arteriosclerosis obliterans are listed in Table 5–6 according to the severity of their symptoms. Interestingly, there is little difference between the mean values of diabetic and nondiabetic patients. Spuriously high pressures due to arterial calcification (which is common in diabetics) seldom occur at toe level. For this reason, toe indices are a reliable indicator of the physiologic severity of arterial occlusive disease and should be used when there is any doubt about the validity of the ankle pressure.[243]

Toe pressures are particularly valuable for recognizing arterial disease confined to the pedal or digital arteries.[77] In limbs with ischemic ulcers or gangrene, normal ankle pressures and normal ankle pressure indices are often associated with toe pressures that lie in the ischemic range (Fig. 5–7).[104, 183]

Normally, toe pressures average 24 ± 7 to 41 ± 17 mmHg less than ankle pressures.[18, 43, 162] Ankle to toe gradients that exceed 44 mmHg in young patients or 64 mmHg in older patients are abnormal.[162] In our experience, the toe-ankle index (obtained by dividing the toe pressure by the ipsilateral ankle pressure) averaged 0.64 ± 0.20 in asymptomatic limbs, 0.52 ± 0.20 in claudicating limbs, and 0.23 ± 0.19 in limbs with ischemic rest pain or ulcers.[183] This suggests that obstruction of the pedal or digital arteries plays a major role in causing gangrene or ischemic rest pain.[243]

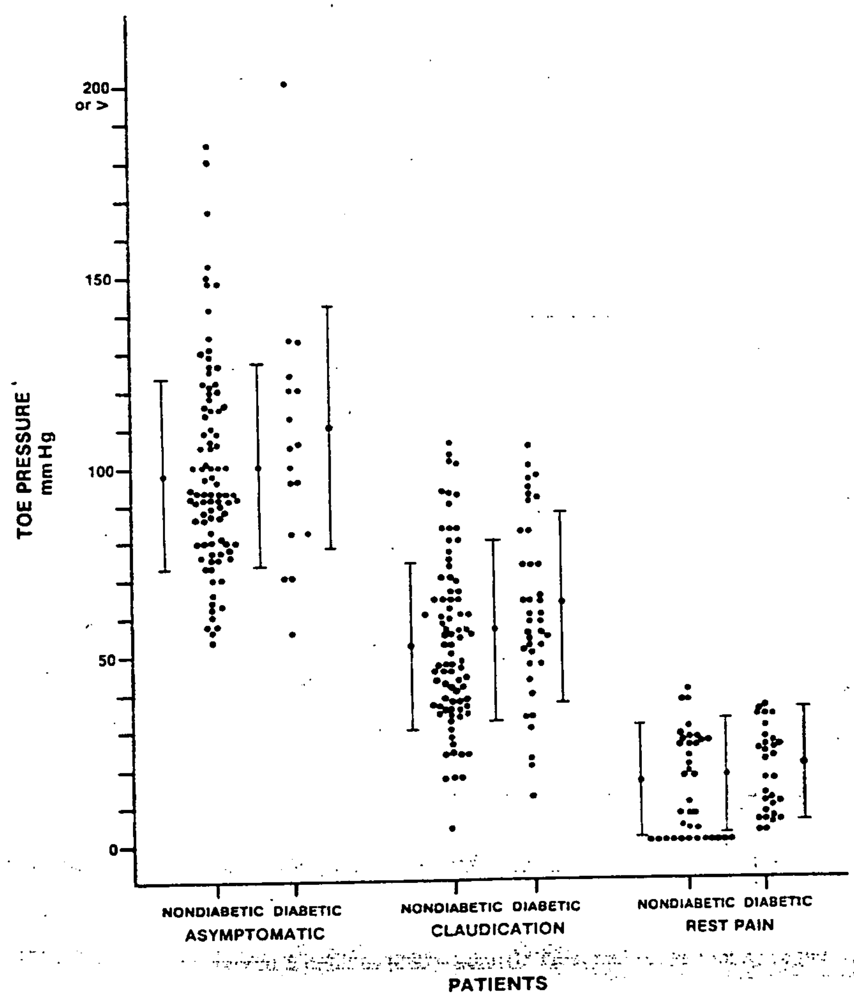


Figure 5–6. Toe blood pressures grouped according to symptoms and presence of diabetes in patients with arterial disease. Mean and standard deviations for the nondiabetic and diabetic subgroups and for the two groups combined are indicated by vertical bars. (From Ramsey DE, Manke DA, Sumner DS: J Cardiovasc Surg *24*:43, 1983.)

Table 5–6. Toe Indices in Patients with Arterial Disease (mean ± SD)

		Claudication		Ischemia*	
Author and Year	**No Symptoms**	*Nondiabetic*	*Diabetic*	*Nondiabetic*	*Diabetic*
Carter and Lezack (1971)[43]	0.91 ± 0.13†	0.43 ± 0.17	0.42 ± 0.16	0.24 ± 0.14	0.19 ± 0.10
Vollrath et al. (1980)[243]	0.89 ± 0.16	0.47 ± 0.24	0.60 ± 0.17	0.19 ± 0.15	0.16 ± 0.13‡
Ramsey et al. (1982)[183]	0.72 ± 0.19	0.35 ± 0.15	0.38 ± 0.15	0.11 ± 0.10	0.12 ± 0.09

*Ischemic rest pain, ulcers, or gangrene.
†Normal subjects, 52 ± 6 years old; 21 ± 4 years old: 0.86 ± 0.12.
‡Diet-controlled; insulin-dependent: 0.23 ± 0.15.

The use of toe pressures for predicting healing of foot lesions or amputations is discussed later in this chapter.

Penile Pressure

The penis is supplied by three paired arteries: the dorsal penile, the cavernosal (deep corporal), and the urethral (spongiosal) arteries. These vessels are terminal branches of the internal pudendal artery, which originates from the internal iliac, or hypogastric, artery. The cavernosal artery is most important for erectile function. Obstruction of any of the arteries leading to the corpora cavernosa—including the common iliac artery or terminal aorta—can be responsible for impotence.

A pneumatic cuff measuring 2.5 cm in width is applied to the base of the penis. Return of blood flow when the cuff is deflated can be detected by a mercury strain-gauge plethysmograph, a photoplethysmograph applied to the anterolateral aspect of the shaft, or a Doppler flow probe.* Although some investigators have positioned the probe over the dorsal penile arteries, others have emphasized the importance of detecting flow in the cavernosal artery.† Because the penile blood supply is paired and obstruction may occasionally be limited to only one side, it has been recommended that pressures be measured on both sides of the penis.[181]

In normal men under 40 years of age, the penile-brachial index (penile pressure divided by brachial systolic pressure) was found by Kempczinski to be 0.99 ± 0.15.[125] In other words, in the absence of any arterial disease, the penile and brachial pressures are roughly equivalent. Older men without symptoms of impotence tend to have lower indices.[125] Penile-brachial indices greater than 0.75 to 0.80 are considered compatible with normal erectile function; an index less than 0.60

*See references: mercury strain-gauge plethysmograph, 34; photoplethysmograph, 136; Doppler flow probe, 125.
†See references: dorsal arteries, 178; cavernosal artery, 158.

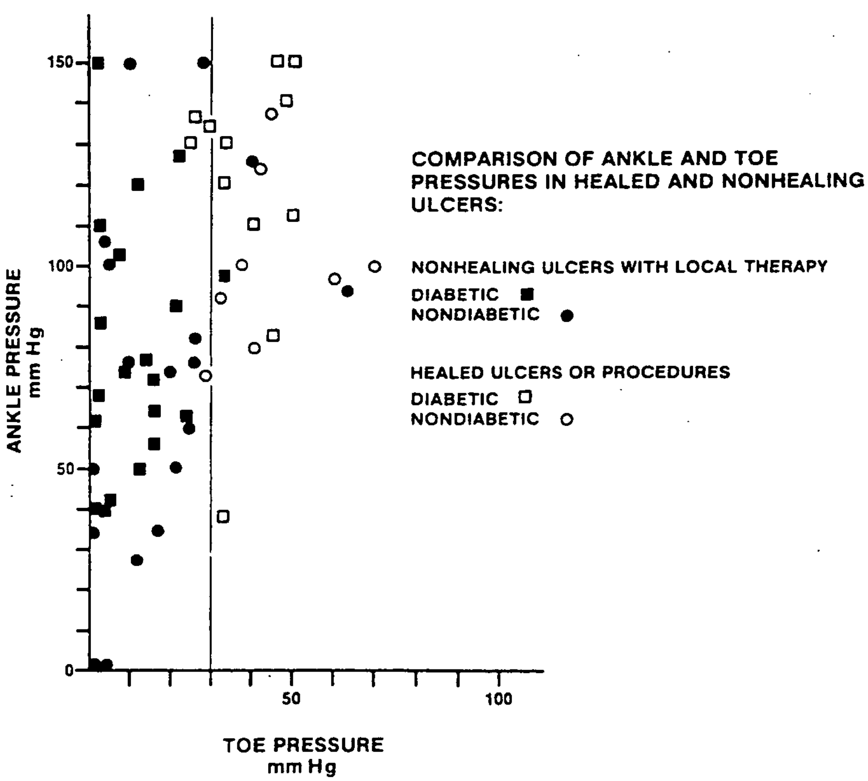


Figure 5–7. Comparison of ankle and toe pressures in 58 limbs with healed or nonhealing ischemic ulcers or toe amputations. Note that a toe pressure of 30 mmHg provides good separation between those limbs that healed and those that did not. Solid symbols indicate nonhealing ulcers: ■ diabetic, ● nondiabetic; open symbols indicate healed ulcers or procedures: □ diabetic, ○ nondiabetic. (From Ramsey DE, Manke DA, Sumner DS: J Cardiovasc Surg *24*:43, 1983.)

is diagnostic of vasculogenic impotence, especially in patients with peripheral vascular disease.[46, 80, 125, 136, 158, 178] A brachial to penile pressure gradient less than 20 to 40 mmHg suggests adequate penile blood flow.[34, 125, 136] Gradients in excess of 60 mmHg suggest arterial insufficiency.[125]

Knowledge of the penile pressure can be used to guide the vascular surgeon in planning the operative approach to aneurysmal or obstructive lesions of the aorta and iliac arteries.[158] Maintenance of blood flow to the internal iliac artery will preserve potency, and restoration of flow to this artery will often improve penile pressure and erectile function.[178]

Stress Testing

Exercise

Reducing the resistance of the peripheral vascular bed by having the patient exercise is an effective physiologic method for stressing the peripheral circulation (see Chapter 3). Under such stress, lesions that may not appear particularly significant at rest can be evaluated.[41] In addition, exercise testing allows the surgeon to better appreciate the functional disability that the arterial obstruction produces.[180] It also permits the surgeon to judge the relative magnitude of the disability produced by arterial obstruction in relation to the restrictions imposed by orthopedic, neurologic, or cardiopulmonary disease.

It should be emphasized that exercise testing (or other stress testing) is not required for the evaluation of patients with ischemia at rest. Patients with ischemic rest pain, ulcers, or gangrene will always have decreased digital artery pressures and will usually have low ankle pressures. Their disease is seldom subtle. Moreover, the vast majority of patients with claudication will have a decreased ankle pressure at rest; consequently, supplementary stress testing is only occasionally necessary to establish the diagnosis.[172] Nevertheless, exercise testing is indicated in certain selected patients for the reasons mentioned in the preceding paragraph. Therefore, it has a restricted but important role in the diagnostic armamentarium.

While many different programs are possible, the following has proved reliable in our hands. After the patient has rested supine for about 20 minutes, a baseline ankle pressure is obtained. The patient then walks on a treadmill at 2 mph up a 10 per cent grade for 5 minutes or until forced to stop because of claudication (or because of other restrictions). The time at which symptoms appear in the leg is noted as well as which muscle group is first affected. In addition, the final walking time is recorded.[224, 251]

The patient then promptly assumes a supine position on the examining table. Ankle and arm pressures are obtained immediately and every 2 minutes until pre-examination levels are reached or until 20 minutes have elapsed. A normal individual, regardless of age, usually will be able to walk for 5 minutes and will experience little or no drop in ankle pressure.[129, 207, 210, 215, 251] Patients with obstructive arterial disease seldom will be able to walk for 5 minutes and will always experience a drop in ankle pressure (Fig. 5–8; see also Figs. 3–14 to 3–17).[215, 224, 251] The magnitude of this drop reflects the extent of the functional disability. Patients with multilevel arterial disease usually will walk for a shorter distance and will experience a much more profound drop in ankle pressure.[251, 255] Often, the ankle pressure will be unobtainable for several minutes (see Figs. 3–16 and 3–17).

Brachial systolic pressure increases after exercise. The increase in pressure is usually much more pronounced in patients with arterial disease than it is in normal subjects. Although an occasional patient with minimal or no symptoms may not demonstrate a distinct decrease in ankle pressure following exercise, the arm to ankle gradient will be increased.[232] Arterial disease may be diagnosed when the postexercise arm pressure exceeds the ankle pressure by more than 20 mmHg. It is rare for such patients to require arterial reconstruction for claudication.

Location of the disease also has an effect on the magnitude of the pressure drop and the time required for the pressure to return to baseline levels (see Fig. 3–14). Pressure drops following exercise indicate that the obstruction involves arteries supplying the gastrocnemius and soleus muscles. Because a large portion of the blood supply to these muscles is derived from the sural arteries, which have their origin from the popliteal, a drop in ankle pressure following exercise signifies an obstruction of the upper popliteal, superficial femoral, or more proximal vessel. When the obstruction is confined to below-knee vessels, exercise seldom causes claudication or a significant drop in ankle pressure—in fact, the pressure may even rise.[215, 216]

In general, the more proximal the occlusive disease is, the more effect it has on the ankle pressure response to exercise. For example, an isolated aorto-iliac lesion usually has more functional significance

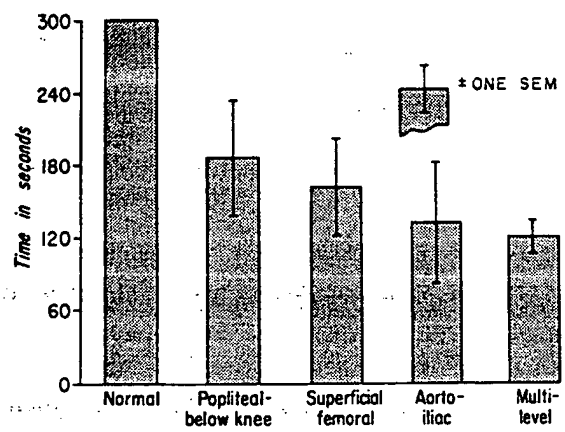


Figure 5–8. Treadmill walking times in patients with occlusive arterial disease. Normal subjects almost always will be able to exceed 5 minutes (300 seconds). Treadmill set at 2 mph, 12 per cent grade. (From Strandness DE Jr, Sumner DS: Hemodynamics for Surgeons. New York, Grune & Stratton, 1975.)

than a lesion confined to the superficial femoral artery.[224, 255] This phenomenon occurs because the more proximal arteries supply a greater muscle mass than the distally located arteries do. Consequently, there is a more severe and prolonged diversion of blood away from the ankle to the proximal muscle masses (see Chapter 3).[220, 224]

Walking time itself is not a particularly important indicator because it is not very reproducible.[172] Motivation, pain tolerance, and ancillary symptoms dictate its duration. It correlates poorly with estimated walking tolerance and with objective hemodynamic measurements.

Reactive Hyperemia

Reactive hyperemia, by increasing the rate of blood flow through stenotic arteries or high-resistance collateral vessels, causes a drop in the ankle pressure similar to that observed after exercise (see Chapter 3).[6, 107, 110, 240] A pneumatic cuff, placed around the thigh, is inflated well above systolic pressure for 3 to 7 minutes. After release of the compression, ankle pressures are monitored at 15-, 20-, or 30-second intervals for 3 to 6 minutes or until measurements return to preocclusion levels. In normal limbs, ankle pressures immediately decrease to about 80 per cent of preocclusion levels but rapidly rise, reaching 90 per cent levels within about 30 to 60 seconds. In limbs with obstructive arterial disease, the decrease in pressure coincides well with that seen following exercise, but recovery to resting levels is much faster.[6, 107] The magnitude of the pressure drop depends upon the anatomic extent of the disease process and on the degree of functional impairment.[110, 240] Although recovery times are also correlated with the severity of the disease (from less than 1 minute to more than 3 minutes), the correlation is not as good as that given by the maximal depression of the ankle pressure.[172]

In some laboratories, reactive hyperemia has supplanted treadmill exercise for stress testing.[107] Compared with treadmill exercise, the test is less time consuming, can be done in the patient's room, and uses simple inexpensive equipment. Since the duration of cuff occlusion can be prescribed and walking time cannot, the stress may be more standardized than that afforded by exercise testing. It is less dependent upon patient motivation. Another frequently cited reason for preferring reactive hyperemia is that it can be used on patients who cannot walk on the treadmill because of neurologic, cardiac, pulmonary, or orthopedic problems or because of general disability, prior amputation, rest pain, or ischemic ulceration.

To me these last arguments are not very convincing. Stress testing is not required to diagnose arterial disease in limbs with rest pain, ulcers, or gangrene. Disease of that severity is easily detected and evaluated with ankle or toe pressures.[172] There is no need to seek out occult disease. In those who cannot walk because of other problems, disease of sufficient severity to jeopardize the limb is readily detected without stress testing; and in such patients, arterial reconstruction for low-grade disease would not be justified. Finally, exercise duplicates the stress responsible for claudication (reactive hyperemia does not) and permits neurologic, cardiopulmonary, and orthopedic problems to be evaluated vis-à-vis the arterial disease.[107]

Some disadvantages of using the reactive hyperemia response can be listed. The test causes mild to moderate discomfort, thigh compression may be hazardous in limbs with femoropopliteal grafts, and rapid pressure measurements are required to get reproducible results.[6, 107, 172] Ouriel and coworkers found that reactive hyperemia was a less sensitive and less specific indicator than resting ankle pressures or exercise tests.[172] Still, the method has some good points, and its use may occasionally be justified.

Doppler Ultrasonography

Although the absolute magnitude of blood flow measured at rest is of little help in the diagnosis or objective assessment of peripheral arterial obstructive disease, the contour of the velocity pulse wave and disturbances of the flow pattern in individual arteries provide a great deal of important information. Before the development of transcutaneous Doppler ultrasonography, this information was essentially unavailable. The presence of a bruit signifies flow disturbance; however, bruits are difficult to quantify, do not appear until the arterial lumen is significantly narrowed, disappear when the stenosis is very severe, and are absent when the artery is totally occluded. Moreover, bruits may arise from arteries adjacent to the vessel of interest, causing additional confusion. Although the electromagnetic flowmeter is capable of displaying the contour of the velocity pulse, it can only be used on exposed vessels, and even then it does not furnish much information regarding subtle flow disturbances. As discussed in Chapter 4, the transcutaneous electromagnetic flowmeter has not filled this void. The noninvasive assessment of arterial blood flow with magnetic resonance is being evaluated, but for many reasons it is unlikely that this modality will soon assume an important role. Laser Doppler flowmetry is applicable only to the cutaneous tissues and cannot be quantified.

Doppler ultrasonography has, therefore, become an essential part of the noninvasive evaluation of peripheral arterial disease. The instruments are not only rugged and easy to use but also provide instantaneous information. Many levels of data analysis are available, ranging from the simple to extremely complex.

Examination Technique

For most purposes, a pencil-type probe is preferred. Optimal signals are obtained by placing the probe directly over the vessel to be examined at an angle of 45 to 60 degrees. In the lower limb, the common femoral artery is examined at the groin at or slightly above the skinfold to avoid confusion with signals

arising from the profunda femoris or the proximal superficial femoral artery. Persson has emphasized the importance of accurately locating the common femoral artery, using the line drawn between the anterior-superior iliac spine and the pubic tubercle to determine the site of the inguinal ligament.[175] The inguinal skin crease, especially in obese patients, is often well below the inguinal ligament.

Signals from the superficial femoral artery are best detected with the probe postioned medially on the thigh in the groove between the quadriceps and adductor muscle bellies. When the patient is supine, flexion of the knee and mild external rotation of the leg provide access to the popliteal artery. Alternatively, the popliteal artery can be examined with the patient prone, the feet being supported by a pillow to flex the knee. At the ankle level, the posterior tibial arterial signal is obtained just behind the medial malleolus. The dorsalis pedis is consistently located slightly lateral to the extensor hallucis longus tendon a centimeter or so distal to the ankle joint. Finally, the lateral tarsal artery (representing the termination of the peroneal artery), can usually be studied by placing the probe anterior and medial to the lateral malleolus over the navicular bone.

Although these represent the sites incorporated in the routine diagnostic evaluation, virtually the entire length of the arteries of the leg can be examined provided the patient's limb is not too large. For example, the posterior tibial and anterior tibial arteries are usually readily detected in mid-calf in their respective anatomic positions, medial and posterior to the tibia and toward the middle of the anterior compartment. The peroneal artery is more difficult to study but can sometimes be located by placing the probe posterior and medial to the fibula. Examination of these more deeply situated arteries is greatly facilitated by the use of a duplex scanner, which allows accurate identification of the vessel and permits precise evaluation of flow.

Although simple nondirectional devices suffice for many clinical applications, direction-sensing instruments supply more information and are necessary for any detailed analysis of the Doppler signal. Even in routine surveys of the peripheral arteries, direction sensing is often a valuable adjunct. The choice of frequency depends upon the depth of the vessel being examined. Whereas superficial vessels are best studied with a high frequency probe (10 MHz), the deeper vessels of the leg require the use of lower frequencies (5 MHz). A 3-MHz probe may be necessary to adequately evaluate flow in the aorta, iliac arteries, and mesenteric vessels.

Audible Interpretation

The ear serves as the simplest and most readily available means of interpreting the output of the Doppler flowmeter. Skilled observers can derive a great deal of information from the audible signal without resorting to recordings or complex methods of analysis. Since good quality, continuous-wave (c-w), nondirectional devices meet most of the requirements for audible interpretation, there is no need for bulky, expensive instrumentation. For many purposes, a hand-held Doppler flowmeter suffices.

Normal arterial signals are biphasic or triphasic.[218] The first sound corresponds to the large, high velocity, forward flow component of the pulse wave; the second, to the smaller reversed flow component in early diastole; and the third, to the even smaller, low velocity, forward flow component that usually appears in late diastole. The pitch of the signal rises rapidly to a peak during systole and then falls abruptly in early diastole. The pitch of the two subsequent signals is always much lower. Finding a clear, crisp, multiphasic signal with a high systolic velocity implies patency of the proximal arteries and almost invariably rules out hemodynamically significant disease.

The characteristics of abnormal Doppler flow signals vary depending on whether the probe is positioned above, at, or well below the site of the occlusive process. Distal to a stenosis or a total occlusion, flow signals are typically low pitched and monophasic, the high frequency components of the pulse wave having been filtered out by passage through the stenosis or high resistance collateral channels. As long as the velocity of flow exceeds a certain minimal level (determined by the transmission frequency and the cut-off frequency of the high pass filter used to eliminate extraneous signals arising from wall motion), arterial signals will be obtained despite the absence of palpable pulses. Absence of a signal implies either a flow velocity below the threshold level or total occlusion of the arterial segment being evaluated. In cases of severe arterial obstruction, the Doppler signal may lose much of its characteristic pulsatility and be difficult to distinguish from an adjacent venous signal. A directional Doppler will usually resolve this issue.

Signals detected over a stenosis or from an artery immediately below a stenosis are high-pitched, noisy, and monophasic. These characteristics reflect the increased velocity of flow within the narrowed lumen and the development of disturbed or turbulent flow patterns in the jet of blood emerging from the stenosis.

Signals obtained from a pulsating artery a few centimeters proximal to a total occlusion have a characteristic "to and fro" or "thumping" quality. This sound is composed of a low frequency forward flow wave followed by a relatively large flow wave reflected from the obstruction. In questionable cases, a directional instrument equipped with frequency meters may aid in the interpretation of the audible signal. When the artery is obstructed distal to the probe and there are no intervening branches to provide outflow, the meters will indicate no mean forward flow or low velocity flow of equal magnitude in both the forward and reversed channels.

Waveform Analysis

The main drawback to the audible interpretation of the Doppler signal is its inherent subjectivity. Waveform analysis is not only objective but it also permits

more information to be extracted from the Doppler shifted signal. As discussed in Chapter 4, several methods are available for processing and recording the velocity signal. Although the zero-crossing output is simple to use, it is often inaccurate and, consequently, is seldom suitable for quantitative work. It does, however, provide a "quick and dirty" method of examining the contour of the waveform, especially in conjunction with segmental pressure measurements. For all serious work, spectral analysis of the audible signal is the method of choice.

Qualitative Analysis

Contour

Simply inspecting the contour of the waveform obtained from the zero-crosser or audiofrequency spectrum often is of considerable diagnostic value. As illustrated in Figure 5–9, the normal velocity waveform is triphasic. Velocity increases rapidly in early systole, reaches a peak, and then drops almost equally as rapidly, reversing in early diastole.[118] In late diastole, the velocity tracing again becomes positive before returning to the zero-flow baseline. With increasing peripheral vasoconstriction, the reversed flow component becomes more exaggerated.[189, 220] When peripheral resistance is reduced following exercise, artificially induced reactive hyperemia, or infusion of vasodilating drugs, the reverse flow component disappears, the baseline rises above the zero-flow level, and the wave assumes a biphasic rather than a triphasic contour. To maintain the characteristics of the normal waveform, recordings should be made in a warm room with the patient resting comfortably in a supine position.

Atherosclerotic changes in arteries proximal to the site of the probe initially produce a disturbance of the contour of the forward flow wave at the peak or in the early deceleration phase. With increasing stenosis, the reversed flow component is dampened and then disappears entirely. As the stenosis becomes more severe, progressing to total occlusion, the rate of acceleration of the forward flow decreases, the peak becomes rounded, and the wave becomes continuous and less pulsatile.[220]

Above a stenosis or occlusion, the wave may have a nearly normal contour, especially when the disease process is located well below the site being evaluated and when there are large outflow branches that serve to reduce the peripheral resistance. It is not uncommon, however, to find that the contour is modified perceptively by increased input impedance and that the resulting wave takes on some of the characteristics commonly associated with proximal stenosis.[161] For example, recordings made from common femoral arteries proximal to superficial femoral occlusions often resemble the waves in Figure 5–9*C* and *D*, even in the absence of any significant iliac stenosis. This must always be kept in mind when one attempts to use the contour of the common femoral waveform to rule in or out inflow disease.

By comparing the contours of the Doppler waveforms obtained from the common femoral, popliteal, and pedal arteries, one can usually identify the presence of hemodynamically significant disease and can often localize the disease to the aortoiliac, superficial femoral, or below-knee segment. The presence of multilevel disease is implied when severely dampened waveforms, such as those shown in Figure 5–9*F*, *G*, and *H*, are recorded from the pedal arteries. Absence of a recordable signal from any of the pedal arteries is indicative of severe ischemia.

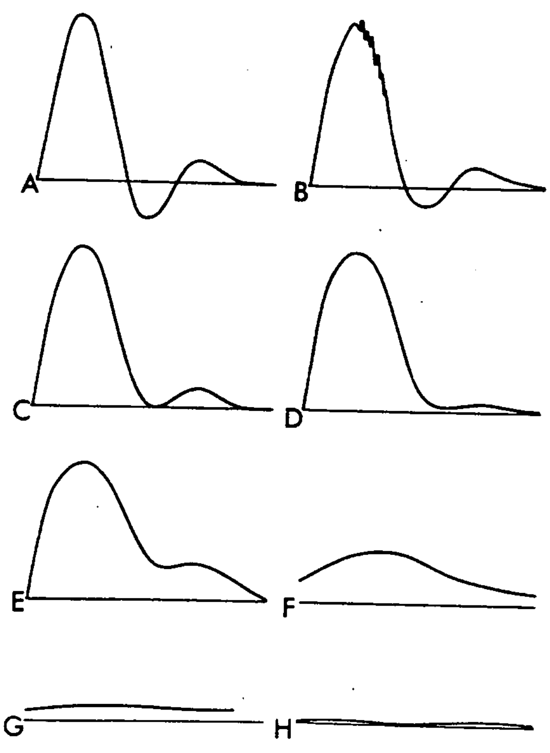


Figure 5–9. Different patterns of flow velocity waveforms. *A*, Normal. *B*, Atherosclerotic changes of artery causing turbulence during systolic phase (high frequency). *C* and *D*, Loss of reverse flow due to progression of degree of stenosis. *E* to *H*, With increasing arterial stenosis, the flow velocity waveform becomes progressively damped. (From Johnston KW, Maruzzo BC, Kassam M, et al: Methods for obtaining, processing and quantifying Doppler blood velocity waveforms. *In* Nicolaides AN, Yao JST (eds): Investigation of Vascular Disorders, London, Churchill Livingstone, 1981, p 543.)

Frequency Spectrum

More precise information can be obtained by surveying the arteries of the abdomen and lower extremity with the duplex scanner.[109, 130, 260] The B-mode image permits the sample volume of the pulsed-Doppler to be placed in the vicinity of the suspected arterial lesion. Thus, frequency spectra are obtained at the site of maximal flow disturbance, not several diameters downstream from the diseased segment, where waveforms may be normalized.[130]

On the basis of the pulsed-Doppler spectrum, stenoses are classified into five categories: *normal,* no diameter reduction; *mild,* 1 to 19 per cent diameter reduction; *moderate,* 20 to 49 per cent diameter reduction; *hemodynamically significant,* 50 to 99 per cent

diameter reduction; and *total occlusion* (Fig. 5–10).[130, 260] The normal spectrum is triphasic (occasionally biphasic) and has minimal spectral broadening (see Chapter 4). A diameter reduction of 1 to 19 per cent is characterized by spectral broadening and loss of the "systolic window." The contour of the wave remains normal, and there is no perceptible increase in peak systolic velocity. Stenoses of 20 to 49 per cent are associated with marked broadening of the spectrum and a 30 to 100 per cent increase in peak systolic velocity compared with the velocity recorded just proximal to the lesion. The reversed flow component is maintained and waveforms proximal and distal to the lesion remain normal. When the diameter stenosis exceeds 50 per cent, the reversed flow component is lost, spectral broadening is marked, and the peak systolic velocity is usually increased by 100 per cent or more. When the stenosis exceeds 80 per cent, the peak velocity of the waveform proximal to the lesion is reduced and the contour becomes monophasic. Distal to these severe stenoses, the contour remains monophasic and spectrum broadening persists. Absence of flow in a clearly imaged vessel is indicative of total occlusion.

Kohler and associates compared the results of spectral analysis and arteriography in 383 arterial segments.[130] For detecting hemodynamically significant stenoses (>50 per cent diameter reduction), the technique had a sensitivity of 82 per cent, a specificity of 92 per cent, a positive predictive value of 80 per cent, and a negative predictive value of 93 per cent. Agreement was perfect in 69 per cent of the studies and was within one category of perfect in 87 per cent. Of particular interest was the 89 per cent sensitivity and 90 per cent specificity for evaluating iliac lesions, which have proved difficult to assess accurately by most noninvasive techniques.

Spectral analysis of Doppler signals obtained during duplex scanning has several advantages: lesions can be accurately localized, multiple lesions can be detected, stenoses can be classified into various degrees of severity, minimal disease can be recognized, and severe stenoses can be distinguished from total occlusion.[130] Moreover, it is possible to study all the major arteries contributing to the circulation of the lower extremity, including the aorta, iliac, common femoral, superficial femoral, popliteal, and profunda femoris, some of which cannot be evaluated by other means.[260] In many patients, it is also feasible to study the anterior tibial, posterior tibial, and peroneal arteries individually. In our laboratory, we have found that real-time color-coding of the flow signal greatly facilitates the examination of the below-knee arteries. The method, however, has some drawbacks. It requires expensive, sophisticated instrumentation, the services of skilled technologists, and approximately 1 hour to thoroughly examine a single leg.[130]

Quantitative Analysis

As discussed in Chapter 4, several methods have been proposed for quantitating the Doppler flow signal.

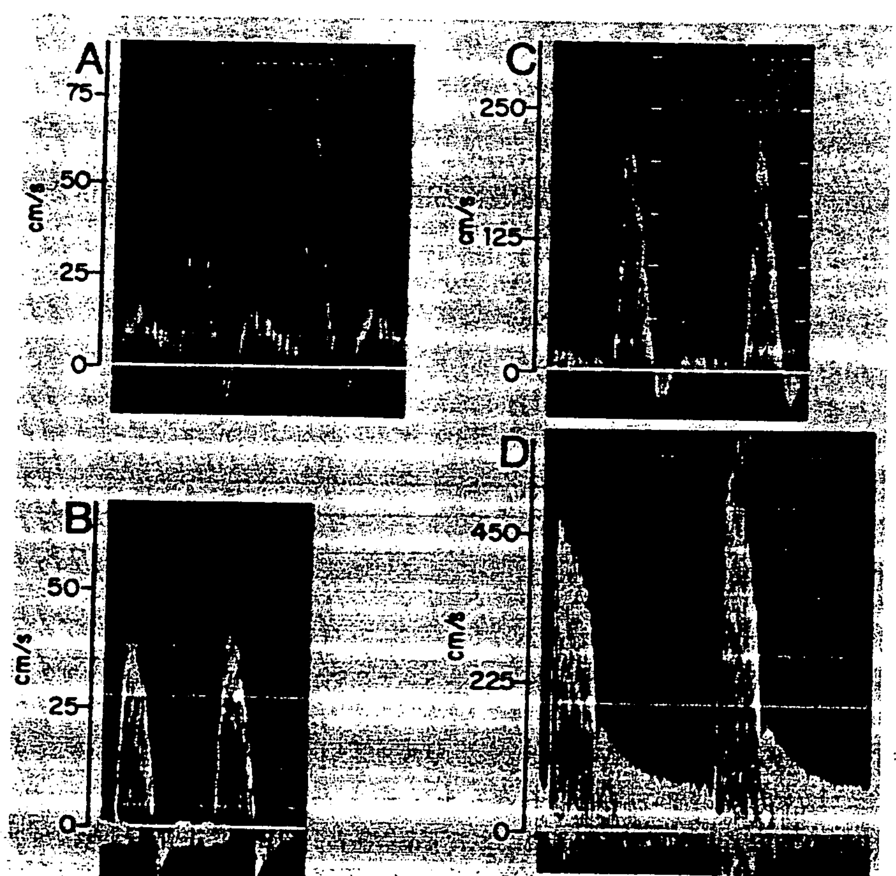


Figure 5–10. Typical velocity spectra for four categories of stenosis: *A*, normal; *B*, 1 to 19 per cent diameter reduction; *C*, 20 to 49 per cent diameter reduction; and *D*, 50 to 99 per cent diameter reduction. (From Kohler TR, Nance DR, Cramer MM, Vandenburghe N, Strandness DE Jr: Duplex scanning for diagnosis of aortoiliac and femoropopliteal disease: A prospective study. Circulation 76:1074, 1987. By permission of the American Heart Association, Inc.)

Table 5–7. Typical Pulsatility Indices (PI) and Inverse Damping Factors (DF^{-1})*

Recording Site	Location of Arterial Obstruction							
	None		*Aortoiliac*		*Superficial Femoral*		*Aortoiliac and Superficial Femoral*	
	PI	DF^{-1}	PI	DF^{-1}	PI	DF^{-1}	PI	DF^{-1}
Common femoral	13.0		2.4		6.1		3.1	
Popliteal	16.7	1.3	2.7	1.1	4.4	0.7	2.4	0.8
Dorsalis pedis	17.7	1.1	5.6	2.1	5.6	1.3	3.7	1.5
Posterior tibial	18.0		4.6		4.6		3.1	

*Data from Johnston KW, Maruzzo BL, Cobbold RSC: Ultrasound Med Biol 4:209, 1978.

Peak-to-Peak Pulsatility Index

In normal legs, the peak-to-peak pulsatility index (PI_{pp}; see Eq. 4.9) increases as the recording site moves from the proximal to distal portions of the limb, being greatest in the dorsalis pedis and posterior tibial arteries and least in the common femoral artery.[115] When, however, there is an intervening arterial stenosis or occlusion, the PI_{pp} value obtained below the involved segment tends to decrease (Table 5–7). Damping factors (DF; see Eq. 4.10) increase and inverse damping factors (DF^{-1}; see Eq. 4.11) decrease across segments with significant arterial disease (Table 5–7).

The PI_{pp} has been advocated as a method for determining the presence or absence of iliac artery disease. As shown in Table 5–8, mean values obtained by several investigators from the common femoral artery agree reasonably well for the various categories of disease severity; but the standard deviations are large, and data from adjacent categories frequently overlap. When arteriography is used as the gold standard, the sensitivity of the PI_{pp} for identifying diameter reductions of the iliac artery greater than 50 per cent (hemodynamically significant stenoses) varies widely from 41 to 100 per cent, depending on the laboratory making the measurements and the value of the index chosen as the dividing point between positive and negative studies (Table 5–9). Specificities are equally variable. Similar data are reported when the pressure drop across the aortoiliac segment, a physiologic gold standard, is substituted for the arteriographic image (Table 5–10).

In general, assessment of the hemodynamic status of the iliac artery seems to be more accurate in the absence of concomitant disease in the superficial femoral artery.[66, 185] Distal arterial obstruction tends to lower the femoral pulsatility index in limbs with no stenosis or low-grade stenoses of the iliac arteries, thereby increasing the number of false-positive study results and reducing specificity (Table 5–10)[8, 66, 114, 185, 231, 245] Some investigators, however, maintain that distal arterial obstruction has little effect on accuracy.[2, 27, 113, 114, 117]

In theory, simple, objective measurements, such as the PI_{pp}, should be relatively consistent from one laboratory to the next. Since pulsatility indices are independent of heart rate and probe angle, there is no obvious explanation for the observed differences other than biologic variability. It is difficult, therefore, to reconcile the disparate opinions expressed in the literature concerning the value of the PI_{pp} for detecting iliac stenosis.[3, 69, 114, 117, 185] Nonetheless, the assumption that a normal femoral pulsatility index (e.g., > 4.0) probably rules out significant iliac stenosis is consistent with many reports.[231] An abnormal index, on the other hand, must be interpreted cautiously, particularly in the presence of infrainguinal obstructive disease.[231]

The mean popliteal pulsatility index in normal limbs was found by Harris and associates to be 9.3 ± 3.6, with a range of 4 to 20.[94] In limbs with femoropopliteal disease mean indices for stenoses less than 50 per cent are reported to be 5.9 ± 3.2 and 7.7 ± 1.1; for hemodynamically significant stenoses they are 4.7 ± 4.0 and 5.9 ± 1.2, and for total occlusion they are reduced to 1.6 ± 0.9 and 2.3 ± 0.2.[2, 94] Obviously, there is a great deal of overlap between values for different grades of disease, but it is usually possible to distinguish between total occlusion and no stenosis.

Table 5–8. Common Femoral Pulsatility Indices (PI) in Limbs with Aortoiliac Occlusive Disease (mean ± SD)

Author and Year	Severity of Diameter Stenosis				
	Normal	*Minimal*	*Less than 50%*	*Greater than or Equal to 50%*	*Occluded*
Johnston et al. (1978)[115]	9.6 ± 2.8	8.1 ± 2.8	4.9 ± 1.3	2.3 ± 1.2	1.6 ± 0.7
Baker et al. (1984)[8]	8.3 ± 7.9	7.6 ± 5.0	3.9 ± 1.2	2.4 ± 0.8	1.8 ± 1.1
Harris et al. (1974)[94]	7.1 ± 1.8	5.7 ± 3.2		2.8 ± 1.1	1.6 ± 0.9
Ward and Martin (1980)[245]	11.1 ± 5.4	4.1 ± 1.8		3.0 ± 1.0	1.9 ± 0.7
Baird et al. (1980)[3]	11.8 ± 5.3	7.4 ± 1.6			
Aukland and Hurlow				3.6 ± 1.6	
(1982)[2]		6.1 ± 2.3		4.3 ± 2.0	1.3 ± 0.6
Hirai and Schoop (1984)[101]	8.0 ± 2.3	6.1 ± 3.0		3.7 ± 1.6	2.0 ± 0.4

Table 5–9. Accuracy of Femoral Pulsatility Indices (PI) for Detecting ≥50% Diameter Stenoses of the Aortoiliac Arterial Segment

Author and Year	Superficial Femoral Artery	PI Criterion*	Sensitivity (%)	Specificity (%)
Flanigan et al. (1982)[69]	—	2.5	70	81
Baird et al. (1980)[3]	—	3.0	41	55
Baker et al. (1984)[8]	—	3.0	76	81
Johnston et al. (1984)[114]	—	3.0	95	97
Baird et al. (1980)[3]	—	4.0	55	71
Baker et al. (1984)[8]	—	4.0	94	66
Campbell et al. (1984)[38]	—	4.0	92	75
Junger et al. (1984)[117]	Occluded	5.0	90	95
Baker et al. (1984)[8]	—	5.5	100	53
Junger et al. (1984)[117]	Patent	7.6	78	89

*PI below which ≥ 50 per cent stenosis is predicted.

Calculation of damping factors provides somewhat better discrimination. Johnston and coworkers state that an inverse damping factor less than 0.9 suggests superficial femoral occlusion.[115] Aukland and Hurlow report DFs of 0.8 ± 0.1, 1.3 ± 0.2, and 2.1 ± 0.2 for minimal disease, hemodynamically significant stenosis, and total occlusion of the superficial femoral artery, respectively.[2] A DF exceeding 1.0 identifies most superficial femoral occlusions; whereas a DF less than 1.9 is highly specific (95 per cent) for the absence of significant disease.[5]

Pulsatility indices obtained from the dorsalis pedis or posterior tibial arteries at the ankle are probably of little practical value in the assessment of below-knee disease. According to Aukland and Hurlow, the mean index in limbs with patent popliteal-tibial segments (6.5 ± 1.4) did not differ significantly from that in limbs in which these arteries were occluded (4.1 ± 0.8).[2] Although Harris and associates found a significant difference between the pedal indices in normal limbs (8.3 ± 3.1) and limbs with below-knee disease, there was no statistically significant difference between any of the angiographic grades (< 50 per cent stenosis, 3.1 ± 2.9; > 50 per cent stenosis, 1.9 ± 2.4; occlusion, 1.1 ± 0.6).[94] On the other hand, the data of Johnston and coworkers suggest that a DF^{-1} less than 1.0 is indicative of severe popliteal-tibial occlusive disease and that a value greater than 1.0 is consistent with normal arteries or arteries with minimal stenosis.[115]

Laplace Transform

The Laplace transform method of waveform analysis was developed to circumvent some of the problems inherent in the interpretation of pulsatility indices (see Chapter 4). It has evoked the most interest as a method for identifying stenoses of the aortoiliac segment, especially in limbs with multilevel disease. Analysis of the femoral waveform yields three parameters, ω_o, γ, and δ, that theoretically relate to arterial wall stiffness, peripheral impedance, and proximal stenosis, respectively.[203] Typical values for δ are given in Table 5–11. These values, which may range from 0.0 to 1.0, tend to increase with increasing severity of stenosis. Statistically significant differences were noted between the means of all adjacent stenosis categories by the first three investigative teams listed in Table 5–11;[3, 114, 203] and Baker and associates, the last team in the list, reported statistically significant differences between the means for stenoses less than 50 per cent and those greater than 50 per cent.[8] All studies, however, have demonstrated appreciable overlapping of individual values from the various categories of stenosis.

The reported accuracy of Laplace δ values for detecting hemodynamically significant stenoses of the aortoiliac segment (≥ 50 per cent diameter reduction) has generally been quite acceptable, most studies indicating sensitivities and specificities well over 85 per cent (Table 5–12). Although some proponents of the

Table 5–10. Accuracy of Femoral Pulsatility Indices (PI) for Detecting Hemodynamically Significant Pressure Drops Across the Aortoiliac Segment

Author and Year	Critical Pressure Drop (mmHg)	Distal Arteries	PI Criterion*	Sensitivity (%)	Specificity (%)
Flanigan et al. (1982)[69]	5	—	2.5	62	69
Johnston et al. (1983)[113]	10	Patent	5.5	95	100
	10	Occluded	5.3	92	92
Thiele et al. (1983)[231]	10	Patent	4.0	95	82
	10	Occluded	4.0	96	45
	20†	Patent	4.0	92	92
	20†	Occluded	4.0	92	51
Bone (1982)[27]	10%‡	—	4.5	94	100

*PI below which pressure drop exceeding the critical value is predicted.
†Critical pressure drop during papaverine-induced hyperemia.
‡Critical drop in femoral/brachial index after reactive hyperemia.

Table 5–11. Laplace Transform Damping Values (δ) in Limbs with Aortoiliac Occlusive Disease (mean ± SD)

Author and Year	Severity of Diameter Stenosis: Normal	<50%	>50%	Occluded
Skidmore et al. (1980)[203]	0.38	0.50	0.70	0.70
Baird et al. (1980)[3]	0.33 ± 0.06	0.50 ± 0.15	0.78 ± 0.16	0.78 ± 0.16
Johnston et al. (1984)[114]	0.41 ± 0.15	0.53 ± 0.18	0.73 ± 0.21	0.89 ± 0.10
Baker et al. (1984)[8]	0.39	0.62	0.82	0.88

Laplace transform method maintain that the δ value is not influenced by distal impedance,[3, 8, 203] others have noted that, like PI_{pp}, the δ value is also sensitive to the presence of superficial femoral arterial occlusive disease.[114, 117] For example, Junger and coworkers found that clamping surgically exposed superficial femoral arteries increased the δ value by 8 to 38 per cent.[117] As pointed out by Johnston and associates, δ is most affected by superficial femoral arterial occlusion when the aortoiliac segment is normal or minimally stenotic.[114] In limbs without inflow disease but with occluded superficial femoral arteries, δ averaged 0.50 ± 0.18, a figure considerably larger than the normal value of 0.35 ± 0.10. The detrimental effect of distal disease on accuracy is most apparent when the δ value is used to distinguish between normal or minimally diseased aortoiliac arteries and those with less than 50 per cent stenosis, greater than 50 per cent stenosis, or total occlusion—superficial femoral arterial occlusion dropping the sensitivity from 94 to 75 per cent and the specificity from 97 to 86 per cent.[114]

Conclusions regarding the relative accuracy of PI_{pp} and δ values differ. Although some authors feel strongly that δ is the better test,[3, 8] others have found both to be equally good.[114, 117, 142] In fact, receiver operator characteristic (ROC) curves of both tests were identical in the study reported by Junger and associates.[117] The data of Campbell and coworkers suggest that PI_{pp} is the better method for detecting iliac stenosis greater than 50 per cent but that δ is better for identifying stenosis of less than 50 per cent.[38] On the other hand, Junger and coworkers found that PI_{pp} was slightly more accurate than δ for identifying low grade stenoses.[117] If the results of the two tests eventually prove to be comparable, PI_{pp}, which has the advantage of simplicity and ease of calculation, would be the method of choice.

Table 5–12. Accuracy of Laplace Transform for Detecting ≥50% Diameter Stenosis of the Aortoiliac Arterial Segment

Author and Year	Superficial Femoral Artery	δ Criterion*	Sensitivity (%)	Specificity (%)
Baird et al. (1980)[3]	—	0.60	85	84
Baker et al. (1984)[8]	—	0.60	100	93
Campbell et al. (1984)[38]	—	0.55	86	69
Junger et al. (1984)[117]	Patent	0.44	88	92
	Occluded	0.52	75	96
Johnston et al. (1984)[114]	Patent	0.66	99	90
	Occluded	0.70	90	88

*δ above which ≥50% stenosis is predicted.

In contrast to δ, the other parameters, ω_o and γ, have received much less attention. Skidmore and associates found that ω_o was related to the stiffness of the arterial wall, increasing as systemic blood pressure increased;[203] however, Junger and coworkers observed no change in wall stiffness with advancing age, although age would be expected to be associated with increasing wall stiffness.[117] Similarly, Junger's group could not confirm Skidmore and associates' observations concerning the correlation of γ and peripheral impedance, finding that γ did not discriminate between normal limbs and those with superficial femoral arterial occlusions.

A ratio of the ω_o calculated from the femoral waveform and that calculated from the posterior tibial or dorsalis pedis artery has been investigated by Campbell and associates as a method for identifying femoral-popliteal occlusive disease.[37] In their study, almost all limbs with ratios exceeding 1.0 had occlusions in this arterial segment, and the results seemed to be independent of the presence or absence of concomitant aortoiliac disease. Although almost all limbs with normal femoral-popliteal segments had ratios less than 1.0, an appreciable number of limbs with occlusions did also. These researchers attributed the false-negative studies to the presence of well-developed collaterals. The overall sensitivity and specificity for identifying superficial femoral arterial occlusion was 75 per cent and 92 per cent, respectively—an accuracy considerably greater than that achieved by calculating damping factors from PI_{pp}.

Velocity Measurements

Fronek and colleagues have advocated quantitative measurement of flow velocities in the common femoral, posterior tibial, and dorsalis pedis arteries as an adjunctive method for assessing peripheral arterial occlusive disease.[74] By making the recordings with the Doppler probe oriented to obtain the maximum output, they minimize the problem posed by the angle of insonation. Measurements include peak forward and reverse velocity, mean velocity, acceleration, and deceleration. From these, peak velocity/mean velocity and acceleration/deceleration indices are calculated. Selected values are listed in Table 5–13. Although the mean values of most of the measurements in diseased limbs differ significantly from those obtained from normal limbs, and mean values from limbs with multisegmental disease differ significantly from many of those from limbs with localized obstructions, the stan-

Table 5–13. Arterial Flow Velocity Measurements (mean ± SD)*

Parameter	Velocity: Normal	Aortoiliac	Popliteal	Femoral-Popliteal
Peak forward velocity (cm/sec)				
Femoral artery	41 ± 11	26 ± 9†	30 ± 15†	21 ± 11†
Posterior tibial artery	16 ± 10	13 ± 12	13 ± 7	12 ± 8†
Dorsalis pedis artery	17 ± 6	15 ± 6‡	11 ± 9†‡	7 ± 7†
Deceleration (cm/sec²)				
Femoral artery	251 ± 60	123 ± 76†	181 ± 117†‡	91 ± 71†
Posterior tibial artery	130 ± 76	79 ± 62‡	77 ± 90†‡	43 ± 40†
Dorsalis pedis artery	138 ± 54	80 ± 51†‡	72 ± 56†‡	29 ± 21†
Peak velocity/mean velocity				
Femoral artery	4.8 ± 1.6	3.1 ± 1.1†	3.6 ± 0.8†‡	2.7 ± 0.8†
Posterior tibial artery	4.8 ± 2.5	3.0 ± 0.8†‡	2.8 ± 1.1†‡	2.1 ± 0.8†
Dorsalis pedis artery	6.0 ± 4.1	3.4 ± 1.5†‡	2.6 ± 0.9†‡	2.0 ± 0.7†

*From Fronek A, et al: Quantitative ultrasonographic studies of lower extremity flow velocities in health and disease. Circulation *53*:957, 1976. By permission of the American Heart Association, Inc.
†p <0.01 compared with normal.
‡p <0.01 localized versus combined disease.

dard deviations are quite large, suggesting that individual measurements are not sufficiently reliable to have much predictive value.[101] When combined with other tests, however, they may be helpful.

Power Frequency Spectrum Analysis

The Doppler frequency spectrum is usually recorded with time on the horizontal axis and frequency on the vertical axis (see Chapter 4). The power (amplitude) at any given frequency is depicted by a gray scale. An alternative format plots frequency at any selected time during the pulse cycle on the horizontal axis and power at the various frequencies on the vertical axis (see Fig. 4–10). Harward, Bernstein, and Fronek used this format to study the power frequency spectrum of the common femoral Doppler signal at peak systole.[95] After examining several parameters that could be derived from the power frequency spectrum, they concluded that the frequency bandwidth at 50 per cent of the maximal amplitude ($f_{50\%}$) provided the most diagnostic information. This parameter is essentially a measure of "spectral broadening" (see Chapter 4). To obtain maximal $f_{50\%}$ values, recordings were made during postocclusive reactive hyperemia, which augments flow through the femoral artery, enhances flow disturbances, and increases spectral broadening.

Analysis of their data demonstrated that a maximal postocclusion $f_{50\%}$ of 2000 Hz proved to be the best discriminator between a positive and negative test for inflow disease. Values less than 2000 Hz were characteristic of limbs with hemodynamically significant aortoiliac stenosis, whereas values above this level were found in normal limbs and in limbs with less severe aortoiliac stenoses. The presence or absence of superficial femoral artery stenosis had no effect on the results—a most important observation. Retrospectively applied, the test had a 93 per cent sensitivity and specificity for detecting isolated aortoiliac disease and a 93 per cent sensitivity and 88 per cent specificity for identifying the presence or absence of hemodynamically significant aortoiliac stenoses in limbs with multilevel disease.

Although these data are encouraging, prospective studies are required before the utility of the test can be determined.

Reactive Hyperemia

Although resting blood flow in limbs with arterial occlusive disease is ordinarily normal and has no diagnostic value, the capacity to increase blood flow in response to peripheral vasodilation is limited (see Chapter 3). This constitutes the rationale for using reactive hyperemia as a test for the presence of arterial disease. Fronek and associates induce reactive hyperemia by inflating a pneumatic cuff placed below the knee to suprasystolic pressures for 4 minutes.[78] Mean flow velocities in the common femoral artery are recorded with a Doppler probe before cuff inflation and then continuously after cuff deflation until velocities return to baseline values. Two parameters are monitored: the percentage increase in velocity, representing the maximal hyperemic response, and the time required for the velocity to return to 50 per cent of its peak value ($T_{1/2}$). In normal limbs, the maximal velocity increase averages about 210 ± 96 per cent of the resting value, and the $T_{1/2}$ averages about 26 ± 9 seconds.[23] Percentage velocity increases are significantly lower in limbs with localized aortoiliac disease, femoropopliteal obstruction, and multilevel disease, averaging 137 ± 112 per cent, 48 ± 54 per cent, and 43 ± 60 per cent, respectively.[23] Similarly, the $T_{1/2}$ is prolonged to 47 ± 22 seconds, 64 ± 51 seconds, and 41 ± 21 seconds in the same disease categories.[23] In the study reported by Ward and Martin, $T_{1/2}$ proved to be a good method for discriminating between normal iliac segments and those with less than 50 per cent stenosis, more than 50 per cent stenosis, and total occlusion, the corresponding mean values being 15 ±

5, 49 ± 14, 68 ± 33, and 91 ± 22 seconds.[245] Hirai and Schoop, however, found that neither the maximal velocity increase nor the $T_{1/2}$ reliably distinguished between < 50% and > 50% diameter stenosis or occlusion of the iliac artery.[101] Because variances are large, this test is largely of supplementary value.

Pulse Transit Time

As discussed in Chapters 3 and 4, the increased wall thickness and decreased wall compliance associated with advancing age and plaque formation tend to accelerate the velocity with which the arterial pulse wave is transmitted in atherosclerotic vessels. Pulse transit times (TT), however, are often prolonged, owing to the extended length of the pathway provided by the more compliant collateral vessels. Transit times are, therefore, most sensitive to the presence of total occlusions, which are always associated with the development of collateral vessels.[2, 108, 245] Most investigators report little difference between the TTs in normal limbs and those in limbs with patent but severely stenosed arteries. For example, Aukland and Hurlow found that the TT from the common femoral to the popliteal artery averaged 35 ± 9 ms over femoropopliteal segments with less than 50 per cent stenosis, 43 ± 7 ms over segments with more than 50 per cent stenosis, and 68 ± 16 seconds over occluded segments.[2] Ward and Martin, who measured TTs from the R wave of the electrocardiogram to the foot of the common femoral pulse, reported mean values of about 150 ms for normal limbs and limbs with both low grade and severe iliac arterial stenoses.[245] But in limbs with occluded iliac arteries, the mean TT was significantly increased to 230 ms.

In an effort to enhance the accuracy of Doppler studies for detecting disease of the femoropopliteal segment, a number of investigators, following the lead of Gosling, plot damping factors obtained from the PI_{pp}s of the common femoral and popliteal arteries (see Eq. 4.11) against the TTs measured across the same segment.[85] To reduce the possible effects of blood pressure variation and age on TTs, the observed TTs are divided by the average "normal" TT across the same segment in limbs of comparable blood pressure and age. (Gosling has provided tables of normal values for this purpose.[85]) Similarly, the observed damping factor (DF) is divided by the mean DF found in normal subjects across the same segment (DF equals 0.7 for the normal femoropopliteal segment).[85] When a scattergram was constructed by plotting the normalized values of the two parameters (DF_N, TT_N) against one another, Gosling and King observed that values obtained from limbs with patent femoropopliteal arteries fell in a box whose upper limits were defined by a DF_N equal to 1.5 and a TT_N equal to 1.5.[86] Totally occluded arteries had DF_N and TT_N values over 1.2. Severely stenotic arteries also had TT_N values in excess of 1.2 but DF_N values less than 1.2. When the superficial femoral arteries were occluded and the collaterals were short, the values for TT_N were less than 2.5; when collaterals were long, values for TT_N exceeded this. The results of Humphries and coworkers' study were similar but did not achieve the 90 per cent accuracy reported by Gosling.[108] Using a cut-off point of 1.9 for both parameters. Baker and associates reported an excellent specificity of 95 per cent but a sensitivity of only 70 per cent.

Although TTs are reasonably successful as a method for identifying total occlusions, this diagnosis can usually be made clinically. Consequently, the applicability of transit time measurements is limited.

Other Quantitative Methods

Rise time (RT) is defined as the duration of the systolic upswing of the flow velocity waveform and is measured from the foot of the wave to the point at which peak velocity is reached. Below an arterial obstruction, rise time is delayed, reflecting the decreased acceleration of the velocity tracing. Obstructions distal to the recording site have little effect on this parameter. In a small series, Hamilton and associates found that RT measured at the common femoral artery discriminated well between limbs with and without significant iliac stenosis.[92] Humphries and associates reported good results in detecting severe stenosis or occlusion of the superficial femoral artery when the ratio of the RT at the popliteal and common femoral arteries was plotted against the transit time between these two sites.[108] RT proved to be as good as DF for this purpose and was much easier to calculate.

Other combinations of measurements have also been used. Nicolaides and coworkers investigated four parameters measured from the common femoral arterial signal: rise time, deceleration time, peak velocity, and pulsatility index.[161] Used alone, none of the parameters was very accurate for diagnosing aortoiliac stenosis; when the results were subjected to multivariate analysis, the combination predicted more than 10 per cent iliac stenosis accurately in 86 per cent of the limbs. The logit of the probability of disease was, however, developed retrospectively, and no prospective study using this method has been reported to assess its accuracy.

Principal component analysis (PCA) is a mathematical technique requiring the use of a computer that describes the waveform in terms of derived coefficients. It is essentially a form of pattern recognition. Although 32 principal components can be determined from the femoral artery waveform, the majority of the information is conveyed by the first two.[142] Early results with this technique have been promising, the combination of the first and second components effectively segregating limbs into three groups: those with severe aortoiliac disease, those with mild aortoiliac disease plus superficial femoral occlusion, and those with mild disease both proximal and distal to the inguinal ligament.[92, 142] In the study reported by Macpherson and coworkers principal component analysis was distinctly more accurate than either the pulsatility index or Laplace δ.[142]

Comment on Waveform Analysis

The large number of methods that have been devised in an effort to provide meaningful quantitation of the Doppler signal suggests that none has proved to be sufficiently accurate. All initially have evoked enthusiasm, but with further experience, their inadequacies have become apparent. The interplay of the multiple factors that dictate the contour and power content of the flow pulse is indeed complex—so much so that it may be impossible to devise any simple measurement that will afford diagnostic information applicable to all the pathophysiologic situations encountered in clinical practice. Many, despite the aura of accuracy that numbers convey, are little better than simple pattern recognition— and perhaps not as good. A major problem is that most derive their information from sites remote from that of the principal lesion where flow patterns have reverted toward a more normal configuration. For this reason, the direct investigation of the artery at the site of the lesion, where flow disturbances are most marked, appears to hold the most promise.[130]

Plethysmography

Plethysmography remains one of the most valuable noninvasive diagnostic modalities available in the vascular laboratory. As outlined in Chapter 4, a variety of instruments are in use, any one of which can be employed in most situations. All measure the same thing: volume change. Although the division may be somewhat arbitrary, it is convenient to discuss segmental and digital plethysmography separately.

Segmental Plethysmography

Although mercury or indium-gallium strain-gauges are quite sensitive and provide excellent recordings of limb volume change, the air plethysmograph, owing to its rugged construction and ease with which it is used, has become the standard device employed in segmental plethysmography. The impedance plethysmograph, while useful for diagnosing deep venous thrombosis, has not proved to be a reliable tool for studying peripheral arterial disease.

Much of the original work with the air plethysmograph was done by Raines and his colleagues, who called their specific instrument the pulse-volume recorder or PVR.[60, 179] This term has now become almost synonymous with segmental plethysmography. Their approach has been to apply pneumatic cuffs to the upper thigh, calf, and ankle. Larger cuffs are used around the thigh (bladder = 18 × 36 cm) and smaller cuffs around the other two sites (bladder = 12 × 23 cm). The cuffs are inflated to 65 mmHg, a pressure that should require about 400 ± 75 ml of air for the thigh cuff and 75 ± 10 ml of air for the other two. Recordings are then made successively from each site. Measurements may be repeated after the patient has exercised on a treadmill.

Pulse Contour

The normal segmental pulse contour is characterized by a steep upstroke, a sharp systolic peak, a downslope that bows toward the baseline, and a prominent dicrotic wave (Fig. 5–11).[60] Significant occlusive disease in arterial segments proximal to the recording cuff is virtually excluded by the presence of a dicrotic wave. Its absence, however, is of less diagnostic significance. For example during the hyperemic period following exercise, the dicrotic wave, which represents reversed flow, may disappear.[127]

Below an arterial obstruction, the upslope is less steep, the peak becomes rounded and is delayed, the downslope bows away from the baseline, and the dicrotic wave disappears (Fig. 5–11).[60] As the proximal obstruction becomes more severe, the rise and fall times become more nearly equal and the amplitude decreases. A "mildly abnormal" form has been identified, the contour of which lies between normal and distinctly abnormal.[127] This pulse retains the rapid upslope and sharp systolic peak characteristic of the normal form but loses the dicrotic wave. The downslope tends to bow away from the baseline. Arterial occlusions distal to the recording cuff may produce a "mildly abnormal" waveform in limbs with no proximal disease. Deterioration toward a distinctly abnormal wave following exercise indicates the presence of significant proximal obstruction.

Pulse Amplitude

According to Darling, Raines, and their associates, the amplitude of the plethysmographic pulse remains highly reproducible in the individual patient provided constant cuff pressures and volumes are used.[60] Amplitudes, however, vary from patient to patient and are influenced by cardiac stroke volume, blood pressure, blood volume, vasomotor tone, and the size and position of the limb. With progressively severe proximal disease, the pulse amplitude decreases. Pulses may be classified into five categories that combine amplitude and specific features of the wave contour (Table 5–14).[180] Category 1 designates a normal pulse wave, and categories 2 through 5 represent waves associated with increasingly severe obstructive disease. Although the actual volume change with each pulse (DV) is greater in the thigh than it is in the calf, the chart deflection at calf level normally exceeds that at the thigh by 25

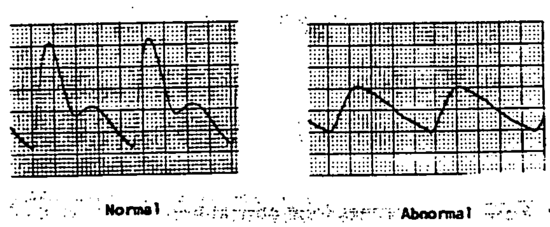


Figure 5–11. Normal and abnormal pulse volume contours recorded at ankle level (cuff pressure, 65 mmHg; cuff volume, 75 ml).

Table 5–14. Definition of Pulse Volume Recorder Categories

Pulse Volume Recorder Category	Chart Deflection (mm)		DV* (cu mm)		
	Thigh and Ankle	*Calf*	*Ankle*	*Calf*	*Thigh*
1	>15†	>20†	>160	>213	>715
2	>15‡	>20‡	>160	>213	>715
3	5–15	5–20	54–160	54–213	240–715
4	<5	<5	<54	<54	<240
5	Flat	Flat	0	0	0

*DV, maximum segmental volume change per heartbeat.
†With reflected wave.
‡No reflected wave.

per cent or more (Table 5–14).[126, 193] This so-called augmentation has proved to be an important diagnostic criterion, its absence signifying the presence of superficial femoral stenosis.

In normal limbs, pulse amplitude increases following treadmill exercise, reflecting the increased blood flow. On the other hand, pulse amplitude at the ankle uniformly diminishes after exercise in limbs with arterial disease, owing to the diversion of blood to the proximal musculature.[179]

Analysis of Pulses

Pulse volume recordings are generally reported to be reasonably accurate for detecting and locating arterial obstructions in the lower extremity. Typical tracings from normal limbs and from those with various combinations of peripheral arterial disease are shown in Figure 5–12.[193] When disease is confined to the aortoiliac segment, pulse contours at all levels are abnormal, but the amplitude of the calf pulse exceeds that of the thigh (a manifestation of the "augmentation" phenomenon). Although pulse contours are also abnormal at all levels when there is combined aortoiliac and superficial femoral arterial disease, the amplitude of the calf pulse is less than that of the thigh pulse. In limbs with isolated superficial femoral arterial obstruction, the thigh pulse is normal but the calf and ankle pulses are abnormal.

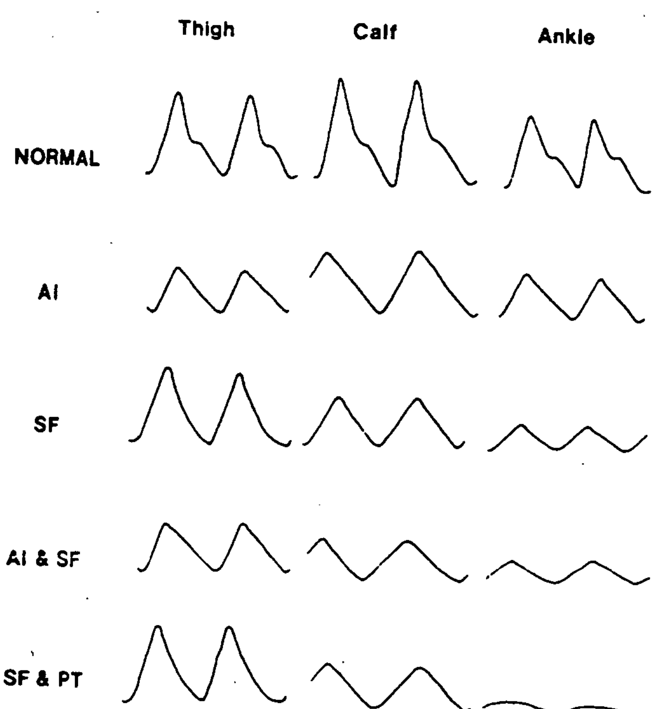


Figure 5–12. PVR tracings from normal limbs and from limbs with various combinations of peripheral vascular disease: AI, aortoiliac; SF, superficial femoral; and PT, popliteal-tibial. (From Rutherford RB, Lowenstein DH, Klein MF: Am J Surg *138*:216, 1979.)

The thigh pulse, according to Kempczinski, tends to underestimate the severity of aortoiliac disease.[126] If, however, moderately abnormal waves were considered to be positive, the PVR correctly identified 95 per cent of the significant stenosis in this segment. There were no false-negatives in his series. All false-positive tests occurred in limbs with stenosis of the profunda femoris artery and occlusions of the superficial femoral segment. Limbs with mildly abnormal thigh pulses were subjected to treadmill exercise at 2 miles per hour up a 10 per cent incline. If the contour of the ankle pulse became more abnormal and if its amplitude decreased, significant aortoiliac obstruction was present; but if there was no change in the pulse, the abnormal thigh pulse was attributed to superficial femoral disease.[126]

The PVR correctly assessed patency of the superficial femoral artery in 97 per cent of the limbs studied by Kempczinski, but pulse changes did not become evident unless the stenosis exceeded 90 per cent diameter reduction.[126] Isolated mid-popliteal occlusions were associated with normal "augmentation" of the calf pulse. All false-positive tests for superficial femoral disease occurred in limbs with aortoiliac disease, and all false-negative tests occurred in limbs with well-developed collaterals bypassing short segmental occlusions of the superficial femoral artery.

Reidy and colleagues reported that the PVR was 100 per cent sensitive to the presence of stenoses of more than 50 per cent of the diameter of the aortoiliac arteries when disease was isolated to that segment; however, the sensitivity fell to 83 per cent in limbs with concomitant superficial femoral arterial occlusions.[186] Although negative predictive values were good for aortoiliac stenoses (87 per cent), positive predictive values were low (64 per cent). Therefore, they considered a positive study to be of little diagnostic value. On the other hand, negative and positive predictive values were quite acceptable (85 and 91 per cent, respectively) when the PVR was used to detect femo-

ropopliteal disease.[126] On the basis of a similar study, Francfort and coworkers concluded that the PVR was inaccurate for detecting aortoiliac disease but that it was highly sensitive to superficial femoral lesions, even in limbs with proximal disease.[72]

Rutherford and associates found that the PVR correctly identified 97 per cent of normal limbs, about 70 per cent of limbs with isolated or combined disease of the aortoiliac and superficial femoral segments, and 100 per cent of limbs with disease confined to the below-knee arteries.[193] When PVR results were considered in conjunction with segmental limb pressures, the accuracy of the combined tests was distinctly better than that of either test alone, ranging between 86 and 100 per cent for all categories of disease. Other investigators have confirmed the complementary roles of the two tests.[72, 126] Therefore, the simultaneous use of PVR and segmental limb pressure measurement is generally advocated. Indeed, this approach was originally recommended by Raines and associates.[180] PVR findings are especially important in subjects with calcified arteries, in whom the segmental systolic pressures are often unreliable.[171]

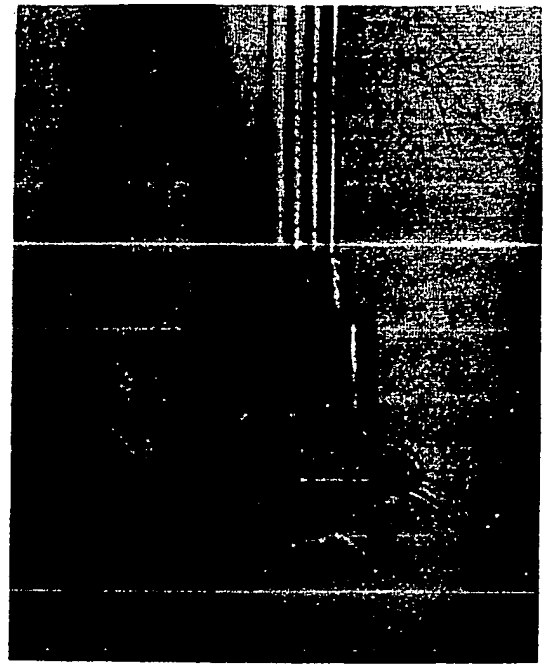


Figure 5–13. A mercury strain-gauge applied to the second toe.

Digital Plethysmography

Although digital plethysmography may be considered a form of segmental plethysmography, pulses obtained from the tips of the toes or fingers (see Chapter 69) have special diagnostic significance. Since the recordings are made from the most distal portion of the extremities, they reflect the physiologic status of all proximal arteries, from the arterioles to the aorta. They are sensitive, therefore, not only to mechanical obstruction but also to vasospasm.

Digit pulses may be recorded with specially designed air plethysmographs that use cuffs with bladders measuring 7 × 2 cm or 9 × 3 cm; however, mercury strain-gauges or photoplethysmographs (PPG) are usually employed because of their greater sensitivity (Fig. 5–13). Although the PPG does not provide quantitative data, it is the most easily used of the three devices and consequently is preferred by many laboratories.

To obtain optimum recordings, studies should be conducted in a warm room (about 72 to 75°F) in which relative humidity is maintained at about 40 per cent. In order to avoid vasospasm, the feet and toes must be warm. This may require immersing the foot in warm water or placing the patient under an electric blanket. At times, it may be necessary to induce postischemic reactive hyperemia (see farther on).[216] While interpretation of pulses of good volume is relatively easy, it is futile and misleading to attempt to assess those of inferior quality.

Pulse Contour

The contour of the digit pulse resembles that of the segmental pulses obtained more proximally in the limb (Fig. 5–14).[52, 211, 216, 250, 259] Normally, there is a rapid upslope, a sharp peak, and a downslope that bows toward the baseline. A dicrotic wave or notch is usually present on the downslope. Distal to an obstruction, the pulse is considerably more rounded, having a slower upslope, a downslope that bows away from the baseline, and no dicrotic wave. In severe cases of arterial obstruction, no pulse may be perceptible.

Finding a normal toe pulse contour is good evidence that all arteries from the digital arteries proximal to the heart are free of functionally significant arterial disease. Similarly, finding an obstructive pulse contour indicates that there are one or more functionally significant areas of obstruction lying somewhere between

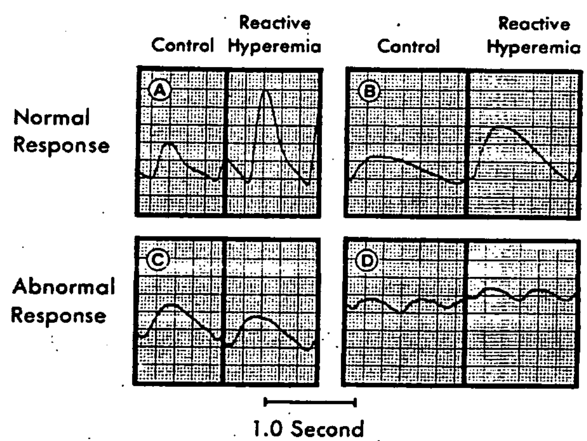


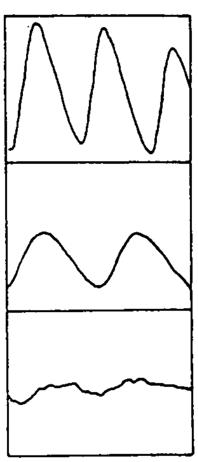
Figure 5–14. Reactive hyperemia test; digit pulse, second toe. Digit pulse volume more than doubles in the normal response *(upper panels)*. Little change in pulse volume is evident in abnormal response *(lower panels.)* *A*, Normal circulation (pressure: arm 130, ankle 140). *B*, Superficial femoral occlusion (pressure: arm 100, ankle 80). *C*, Diabetic for 20 years (pressure: arm 135, ankle 135). *D*, Iliac and superficial femoral arterial disease (pressure: arm 118, ankle 46). Attenuation of recorder: *A* × 10; *B, C, D,* × 20.

the heart and digital arteries. Thus, digital pulses are especially important in the investigation of ischemia of the toes or forefoot.[223] Pedal or digital artery disease, which may contribute greatly to the ischemic process, may escape detection if the investigation is carried no further than the ankle level. Such errors are easily made in diabetic patients, because the ankle arteries are often incompressible and the disease is likely to be concentrated in the pedal vessels. As shown in Figure 5–15, perfusion of the toes may be inadequate even in the presence of a good ankle pressure.

Although more complex methods for describing digital pulse contours have been proposed (including measurements of slope, pulse width at half maximum excursion, and relative amplitudes at various parts of the curve), these measurements have little physiologic meaning and are unnecessary in clinical work.[19, 22]

Pulse Amplitude

The volume of the toe pulse is not in itself a very reliable indicator of the severity of arterial disease.[52, 211, 259] Although the digital pulse volume corresponds quite closely with digital blood flow (provided that the same gauge is used on the same toe of the same subject during the same examination), it is impossible to compare toe pulses from day to day in the same subject or between subjects as an index of relative blood flow.[261] Moreover, digital blood flow is highly variable, depending more on sympathetic activity than on the presence of anatomic arterial disease.



PRESSURE – mmHg TOE	ANKLE	ANKLE INDEX
60	100	0.79
23	60	0.34
13	94	0.51

Figure 5–15. The configuration of the toe plethysmogram is closely correlated with the toe pressure but is poorly correlated with the ankle pressure or ankle index. Upper tracing implies good digital artery perfusion; middle tracing, borderline perfusion; and lower tracing, ischemia. (From Sumner DS: Rational use of noninvasive tests in designing a therapeutic approach to severe arterial disease of the legs. *In* Puel P, Boccalon H, Enjalbert A (eds); Hemodynamics of the Limbs-2, Toulouse, France, GEPESC, 1981, pp 369–376.)

Table 5–15. Pulse Reappearance Time After Release of Arterial Occlusion (seconds)*

Location of Occlusive Disease	Pulse Reappearance Time	Time Required to Reach Half Control Volume
No occlusions	0.2 ± 0.1	3.4 ± 0.8
Aortoiliac	7.2 ± 4.0	23.9 ± 6.7
Femoropopliteal	3.7 ± 3.7	26.5 ± 12.7
Popliteal trifurcation	15.2 ± 9.3	23.9 ± 9.4
Multilevel	45.3 ± 5.5	71.2 ± 5.5

*Modified from Fronek A, Coel M, Bernstein EF: Surgery *81*:376, 1977.

Reactive Hyperemia

The reactive hyperemia test is valuable as an indicator of the extent of peripheral vascular disease and as a predictor of the efficacy of surgical sympathectomy.[209, 216] A pneumatic cuff is placed around the ankle, calf, or thigh; inflated to above systolic pressure; kept at that level for 3 to 5 minutes; and rapidly deflated. The volume of the toe pulse is then followed over the next several minutes. In normal limbs, the pulse returns almost immediately, attains half its preocclusive amplitude within a few seconds, and then rapidly reaches a peak volume (Table 5–15).[75, 90, 223] Maximum excursion is usually more than twice that recorded during the control period (see Fig. 5–14).

The reappearance time of the toe pulse is frequently delayed in legs with arterial occlusive disease, often exceeding 120 seconds in severely impaired extremities.[75, 90] Because it is difficult to define precisely the time at which the first pulse returns, the time required for the pulse to reach half the preocclusive volume seems to be a more practical measurement. This value is closely related to the severity of the disease and to the extent of arterial occlusion (Table 5–15). Bernstein and colleagues have found that the functional results of aortofemoral bypass procedures can be predicted quite well by determining the time required for the pulse to reach half the control volume.[24] When the time was less than 10 seconds, 63 per cent of the limbs became asymptomatic and another 37 per cent were improved. However, when the time exceeded 90 seconds, only 10 per cent of the limbs became asymptomatic and only 50 per cent improved.

The relative increase in pulse volume during reactive hyperemia is an excellent indicator of the functional severity of the arterial obstruction. As discussed in Chapter 3, the peripheral vascular bed is nearly maximally dilated in limbs with severe ischemia; consequently, little further vasodilatation is possible (see Fig. 5–14). Approximately 85 per cent of limbs with obstruction confined to a single segment (e.g., aortoiliac, femoropopliteal, or popliteal trifurcation) display at least a 25 per cent increase in the volume of the toe pulse.[75] In contrast, only about half of the limbs with multilevel disease show a response of this magnitude. Because of proximal steal phenomena, the pulse volume in such limbs may remain decreased for a long period and may never exceed that recorded during the control period.[223]

Experience has shown that surgical sympathectomy is most likely to provide a satisfactory result if the peak reactive hyperemia pulse is twice the size of the control pulse. If less dilatation is seen, it is much less likely that sympathectomy will increase peripheral blood flow. Since reactive hyperemia can be obtained in a fully sympathectomized extremity, the reactive hyperemia test reveals nothing about the integrity of the sympathetic innervation; what the test does is to substantiate the ability of the peripheral vessels to dilate in response to a release of vascular tone. The "deep breath test" or one of its modifications is necessary to demonstrate continued function of the sympathetic nervous system.[64] In response to a deep breath, the pulse volume will temporarily decrease, provided sympathetic innervation is intact. Absence of this response implies impaired sympathetic activity and should be considered a contraindication to sympathectomy.

Transcutaneous Oxygen Tension

Unlike the methods previously discussed in this chapter, which are sensitive to hemodynamic changes, transcutaneous oxygen tension ($tcPo_2$) measurements reflect the metabolic state of the target tissues. Although the technique is susceptible to a host of confounding variables, the potential importance of the information derived is so great that the method deserves close attention.

Although measurements may be obtained from almost any area of interest, common locations include the dorsum of the foot, the anteromedial calf about 10 cm below the patella, the thigh about 10 cm above the patella, and the chest in the subclavicular region. In normal limbs, most—but not all—investigators have observed a modest decrease (5 to 6 mmHg) in $tcPo_2$ from the more proximal parts of the leg to the foot.[36, 47, 145] With increasing age, $tcPo_2$ tends to decrease, paralleling a similar decline in arterial Po_2.[47, 87, 99, 156, 169] For this reason, Hauser and Shoemaker have advocated dividing the limb $tcPo_2$ by that measured at the subclavicular region to obtain a regional perfusion index (RPI) that is independent of age, cardiac output, and arterial Po_2.[99] Others maintain that this calculation does not significantly enhance the predictive value of the test.[36, 47, 156] Values for $tcPo_2$ also depend on the vertical distance between the measurement site and the heart, decreasing when the limb is elevated and increasing when the limb is dependent.[36, 73, 98, 99, 145] In addition, there may be some increase in $tcPo_2$ with an elevation of venous pressure.[73]

As discussed in Chapter 4, $tcPo_2$ values represent a complex function of cutaneous blood flow, metabolic activity, oxyhemoglobin dissociation, and oxygen diffusion though the tissues. Changes are not ordinarily perceptible in limbs with mild degrees of arterial disease, since the oxygen supply far exceeds that required to meet metabolic demands. Under conditions of stress, however, the metabolic demands may encroach on the available oxygen supply, thereby reducing the $tcPo_2$. Likewise, in cases of severe arterial obstruction, oxygen delivery is often marginal; the quantity of free O_2 reaching the sensor is reduced, and the $tcPo_2$ falls. Thus, $tcPo_2$ is most sensitive to the higher grades of arterial obstruction. Even at low levels of perfusion, $tcPo_2$ values are not linearly related to blood flow.[146] In fact, $tcPo_2$ may fall to zero in areas where cutaneous blood flow is still detectable by other methods.[146] This does not mean that no oxygen is reaching the tissue but does imply that all available oxygen is being consumed and that none remains for diffusion to the sensor.[47, 73, 35]

Since the arterial pressure at any given site represents the potential energy available for transporting blood through the tissues, it is not surprising that statistically significant correlations between segmental pressures and $tcPo_2$ values are reported.[48, 145, 169, 253] The correlation coefficients, however, are quite poor, especially in diabetic extremities.

Resting Values

Representative $tcPo_2$ values obtained from resting supine subjects are given in Table 5–16. The tendency for the values to decrease from the more proximal to the more distal parts of the leg is minimal in normal limbs but becomes more pronounced with increasing severity of the disease. Irrespective of the site of measurement, normal $tcPo_2$ hovers around 60 mmHg. Measurements in normal younger subjects are usually about 10 mmHg higher than those given in the table, which correspond to values observed in patients in the age groups most susceptible to atherosclerosis.[47, 99, 156] In general, a $tcPo_2$ greater than 55 mmHg may be considered normal at any measurement site regardless of age.[47] The average normal RPI is about 90 per cent.[99]

Peripheral measurements reflect the deleterious effect of increasing obstructive disease more dramatically than the more proximal measurements do. For example, there is little difference among the thigh $tcPo_2$ values of any of the disease groups listed in Table 5–16. Although statistically significant differences are often demonstrated between the value of normal and claudicating extremities and between claudicating extremities and those with rest pain, there is enough overlap to prevent individual tests from discriminating accurately among the various disease categories.[47] Many claudicants have resting $tcPo_2$ values that fall in the normal range, even at foot level.[36] Values in limb-threatening ischemia are, however, significantly reduced. At foot level, $tcPo_2$ values are usually less than 20 mmHg in legs with severe rest pain, ischemic ulcers, or gangrene.[36, 47, 168] In the series reported by Wyss and coworkers 46 per cent of nondiabetic limbs with $tcPo_2$ values less than 20 mmHg required amputation.[253]

Table 5–16. Representative $tcPo_2$ Values at Rest, Supine Position (mmHg, mean ± SD)

Author and Year	Normal*			Claudication			Rest Pain		
	Foot	*Calf*	*Thigh*	*Foot*	*Calf*	*Thigh*	*Foot*	*Calf*	*Thigh*
Clyne et al. (1982)[48]	59 ± 4	63 ± 5	64 ± 6	51 ± 10	64 ± 9	67 ± 9	36 ± 16	50 ± 16	55 ± 18
Hauser and Shoemaker (1983)[99]	59 ± 10	56 ± 10	64 ± 7	46 ± 12	49 ± 9	57 ± 9	—	—	—
Cina et al. (1984)[47]†	64 ± 4	64 ± 4	—	46 ± 5	55 ± 4	—	17 ± 4	42 ± 6	—
Byrne et al. (1984)[36]	60 ± 7	63 ± 8	66 ± 8	56 ± 4	59 ± 5	66 ± 7	4 ± 4	29 ± 20	50 ± 14
				37 ± 12	48 ± 10	54 ± 7‡			
Kram et al. (1985)[134]	47 ± 12	53 ± 15	—	33 ± 14	37 ± 13	—	20 ± 16	29 ± 20	—

*Older subjects.
†Values estimated from published graphs.
‡More severe claudication.

Enhancement Procedures

Recordings may be made after exercise, following a period of ischemia. during oxygen inhalation, and with the legs in a dependent position. These are among the various modifications of the basic measurement procedure that have been advocated to enhance the discriminatory ability of the $tcPo_2$ values.

Dependent Position

Franzeck and colleagues observed an average increase of 15 ± 7 mmHg in the $tcPo_2$ on the dorsum of the foot in normal subjects when they moved from a supine to a sitting position.[73] In this position, the sensor was 54 cm below the heart. With standing, which extended the distance to 84 cm, the $tcPo_2$ rose by an average of 28 ± 14 mmHg. The increase in $tcPo_2$ that accompanies standing occurs at all levels of the leg but is most evident at foot level, where the hydrostatic pressure is greatest.

As a rule, the augmentation in $tcPo_2$ increases commensurate with the severity of limb ischemia. Byrne and associates, for example, noted an average increase of 20 mmHg in limbs with rest pain compared with an average increase of 10 mmHg in normal limbs.[36] Based on their data, it appears that $tcPo_2$ rises by about 18 per cent in normal limbs, 22 per cent in claudicating limbs with normal resting $tcPo_2$ values, 58 per cent in claudicating limbs with abnormal resting values, and 88 per cent in limbs with rest pain. Oh, working in the same laboratory, retrospectively separated severely ischemic extremities into two groups based on the change in $tcPo_2$ that occurred with standing.[168] Group I was defined by a $tcPo_2$ increase of less than 15 mmHg (average 4 ± 5 mmHg) and group II, by an increase of more than 15 mmHg (average 36 ± 11 mmHg). Despite the fact that both groups had similar supine $tcPo_2$ values (4 ± 5 mmHg and 6 ± 5 mmHg, respectively), the manifestations of disease were more severe in group I limbs (61 per cent ulcers or gangrene, 48 per cent rest pain, and 39 per cent claudication) than in group II limbs (29 per cent ulcers or gangrene, 46 per cent rest pain, and 68 per cent claudication).

The increase in oxygen tension that occurs when a patient with an ischemic limb sits or stands may explain how dependency relieves rest pain. As discussed in Chapter 3, elevation of the hydrostatic pressure dilates capillaries and other resistive vessels, thereby permitting more blood to flow at the same arteriovenous pressure gradient. In addition, any muscular activity with the leg dependent may decrease venous pressure and increase the arteriovenous pressure gradient (see Chapter 132). With the increase in capillary blood flow, more oxygen is delivered to the tissues. In the most severely ischemic extremities, these physiologic buffers are nearly exhausted, and the increase in blood flow is inadequate to provide relief.

Exercise

In limbs with restricted arterial inflow, dilatation of intramuscular vessels induced by exercise diverts blood away from cutaneous vascular beds, causing $tcPo_2$ to fall. In normal limbs, this "steal" is not evident, since the arterial blood supply is adequate to supply both vascular beds. It is not surprising, therefore, to find that postexercise/pre-exercise ratios of ankle pressures and ankle $tcPo_2$ values are highly correlated (r = 0.918), as demonstrated by the treadmill studies of Matsen and associates.[147]

Byrne and coworkers observed that $tcPo_2$ values measured on the dorsum of the foot during treadmill exercise remained at about the same level as those obtained during quiet standing (which is somewhat difficult to explain); however, during the period following exercise, after the subjects had resumed a supine position, the $tcPo_2$ values fell in all patients with significant arterial obstruction, even in those with normal resting values (Table 5–17).[36] No fall was evident

Table 5–17. Effect of Exercise on Foot $tcPo_2$ (mmHg, mean ± SD)*

Type of Obstruction	$tcPo_2$ Values			
	Supine	*Standing*	*Exercise*	*Post-exercise*†
Normal	60 ± 7	71 ± 7	75 ± 9	69 ± 7
Claudication‡	56 ± 4	58 ± 8	53 ± 10	33 ± 16
Claudication	37 ± 12	58 ± 12	49 ± 18	23 ± 20
Rest Pain	4 ± 4	25 ± 20	26 ± 26	5 ± 7

*Data derived from Byrne P, Provan JL, Ameli FM, et al: Ann Surg *200*:159, 1984.
†Postexercise measurements made with patient supine.
‡Claudicants with normal resting $tcPo_2$ values.

in normal subjects. Similar, but less marked, changes occurred at calf level. They concluded that postexercise $tcPo_2$ measurements accurately distinguished all subjects with vascular claudication from those who were normal.

Hauser and Shoemaker found that the chest $tcPo_2$ increased in claudicants during exercise, perhaps explaining the failure of limb values to decrease.[99] However, the RPI (limb $tcPo_2$/chest $tcPo_2$) at foot level did decrease during exercise, even in normal extremities. When RPIs obtained during exercise were compared with values obtained when the patient was standing, a decrease of more than 10 per cent at the thigh or more than 15 per cent at the calf was found to be highly specific for intermittent claudication. Normal limbs measured at these levels demonstrated no fall in RPI. After exercise, the RPI at the foot in normal limbs always returned to pre-exercise values within 1 minute. In claudicating limbs, the average time to recover one half of the exercise drop was about 4 minutes.

Reactive Hyperemia

Inflation of a pneumatic cuff to suprasystolic pressure level is followed by a rapid decline in the $tcPo_2$ measured further down the leg. When the cuff is deflated after a period of ischemia (usually about 4 minutes), the $tcPo_2$ rapidly returns to preocclusion levels in normal extremities. In limbs with occlusive arterial disease the rate of recovery is much slower. Recovery rates are usually expressed as the time required for the $tcPo_2$ to return to one half of the preocclusive value ($T_{1/2}$).

Representative results are shown in Table 5–18. Cina and coworkers reported a range of 43 to 60 seconds in normal limbs and 75 to 150 seconds in claudicating limbs; there were no overlapping values.[47] Kram and associates found that postischemic recovery times based on the RPI (limb $tcPo_2$/chest $tcPo_2$) were more diagnostic of arterial disease than toe-pulse recovery times.[132] Values for $T_{1/2}$ in excess of 84 seconds at the calf and 102 seconds at the foot were considered pathologic.[132, 134]

Oxygen Inhalation

Inhalation of pure oxygen markedly augments the $tcPo_2$ in normal limbs but has less effect in limbs with severe arterial occlusion. Ohgi and associates reported that pretibial values increased from 70 ± 9 to 365 ± 87 mmHg in normal legs, from 34 ± 33 to 115 ± 109 mmHg in legs with chronic occlusions, and from 23 ± 30 to 96 ± 57 mmHg in legs with acute occlusions.[169] According to Harward and associates, the prediction of amputation healing by means of $tcPo_2$ determinations may be enhanced by oxygen inhalation.[96]

Comment on $tcPo_2$ Measurements

Although $tcPo_2$ measurements made after exercise or a period of ischemia can be used to distinguish between normal and claudicating limbs, other less demanding tests are equally efficacious. It would seem that a more appropriate role for $tcPo_2$ measurement is to assist in the assessment of severe ischemia. Since the results are not affected by arterial calcification, this test is particularly valuable for evaluating diabetic vascular disease.[47, 73, 98, 119]

Laser Doppler

A relative index of cutaneous blood flow can be obtained with the laser Doppler (see Chapter 4). The output, which is expressed in millivolts (mv), is roughly proportional to the average blood flow in a 1.5 mm^3 volume of skin, lying 0.8 to 1.5 mm below the skin surface. According to Karanfilian and associates, tracings from normal skin exhibit three major characteristics: (1) pulse waves that coincide with the cardiac cycle, (2) vasomotor waves that occur four to six times per minute, (3) and a mean blood flow velocity that is represented by the elevation of the tracing above a zero baseline.[118] In the leg, the highest velocities are obtained from the skin of the big toe, followed, in descending order, by velocities from the skin of the plantar surface of the foot, dorsal foot, distal leg, thigh, and proximal leg.

In limbs with peripheral vascular disease, pulse waves are attenuated, mean velocities are decreased, and vasomotor waves may disappear (Table 5–19).[119] The reactive hyperemic response to a period of cuff-induced ischemia is diminished, and the time to reach maximum hyperemia is markedly delayed (Table 5–19).[119] Karanfilian and associates investigated the ability of laser Doppler studies to predict healing of ulcers or forefoot amputations in a series of ischemic limbs.[119] When the mean velocity recorded from the plantar aspect of the foot or big toe exceeded 40 mv and the pulse wave amplitude exceeded 4 mv, 96 per cent of the lesions or amputations healed. On the other hand, when these criteria were not met, 79 per cent of the feet failed to heal. These results were not quite as good as those obtained on the same extremities using $tcPo_2$.

Table 5–18. Postischemic Transcutaneous Oxygen Recovery Rate $T_{1/2}$ (seconds)

Author and Year	Normal		Claudication		Rest Pain	
	Foot	Calf	Foot	Calf	Foot	Calf
Franzeck et al. (1982)[73]	87 ± 27	60 ± 15	136 ± 73	131 ± 69	—	—
Cina et al. (1984)[47]	49 ± 6	—	114 ± 2	—	—	—
Kram et al. (1985)[134]*	66 ± 18	48 ± 18	156 ± 60	126 ± 42	204 ± 78	126 ± 66

*Based on limb/chest $tcPo_2$ ratio.

Table 5–19. Laser Doppler Measurements from the Big Toe (mean ± SD)*

	Baseline Values		Reactive Hyperemia Test	
	Velocity (mv)	*Pulse Amplitude (mv)*	*Peak/Baseline† Ratio*	*Time (sec) to Maximal Velocity*
Normal	197 ± 174	77 ± 63	3.1 ± 0.9	18 ± 7
Ischemic	67 ± 42	5 ± 4	1.7 ± 1.6	150 ± 48

*Data derived from Karanfilian RG, Lynch TG, Lee BC, et al: Am Surg *50*:641, 1984.
†Ratio of peak postischemic velocity to preischemic velocity.

The laser Doppler can also be used in conjunction with a pneumatic cuff to estimate skin blood pressure at almost any level of the upper or lower extremities. These measurements are made with the probe (which merely serves as a flow sensor) placed under the pneumatic cuff. Castronuovo and associates obtained cutaneous pressure averaging 47 ± 28 mmHg in normal forearms and thighs and 73 ± 28 mmHg in the plantar skin of the big toe.[45] These pressures are similar to those in precapillary vessels. Much lower values were found in the plantar skin of the toe (17 ± 15 mmHg) and in the dorsal skin of the foot (10 ± 10 mmHg) in limbs with rest pain, ulceration, or gangrene.

Whether the laser Doppler will prove to be a valuable asset to the vascular laboratory remains to be seen. It cannot be calibrated in terms of actual blood flow, and much of the information that it provides is already supplied by more established tests.

Clinical Applications

After the history and physical examination have been completed, the next logical step in the evaluation of a patient for arterial disease is physiologic testing.[19] These tests are designed to answer the following questions:

1. Is significant arterial occlusive disease present?
2. If so, how severe is the physiologic impairment?
3. What is the approximate location of the disease?
4. In multilevel disease, which arterial segments are most severely affected?
5. In limbs with tissue loss, what is the potential for healing?

Armed with this information, the surgeon is better able to decide whether the deficit is severe enough to warrant diagnostic radiology as a prelude to possible surgical intervention.

Some surgeons protest that physiologic testing is unnecessary, provided a careful history and physical examination have been carried out. This argument fails to take into account a number of clinical problems that are not easily resolved even with the help of arteriography. For example, patients with pseudoclaudication may be identified and spared further vascular workup.[44, 84, 123, 230] Patients with demonstrable arterial disease may have concomitant orthopedic or neurologic problems. Often with the help of physiologic testing, the physician can determine the relative magnitude of the deficit caused by each of the diseases and advise the patient accordingly. In limbs with multilevel disease, it is sometimes possible to identify which of the lesions is most significant, allowing the surgeon to focus on the more critical lesion.[3, 24, 31; 161, 175, 227, 245] Stenoses that appear mild on arteriograms frequently cause significant physiologic deficits; others that appear severe may be relatively inconsequential.

Physiologic tests are helpful in determining whether an ulcer is due to neuropathy, stasis, infection, or ischemia and in deciding whether foot pain is primarily neuropathic or ischemic.[106, 183] They may also enhance the ability of the surgeon to assess the healing potential of a foot lesion or amputation and may help in selecting the appropriate site for amputation.[16, 28, 103, 153, 242] In cases of suspected vascular trauma, physiologic findings, if negative, may avert an unnecessary vascular exploration or, if positive, may alert the surgeon to the need for immediate operation. Similarly, the recognition and evaluation of suspected iatrogenic vascular injuries, such as those that follow cardiac catheterization or interventional radiology, are facilitated by physiologic testing.[15] Physiologic tests are uniquely applicable to the diagnosis of intermittent arterial obstructions, such as those arising from entrapment syndromes, and for distinguishing between fixed arterial obstructions and those due to vasospasm. These are but a few of the many areas in which physiologic tests complement the information gleaned from the routine history, physical examination, and arteriogram.

Once all the data have been analyzed, the surgeon must decide between various therapeutic options. No matter what course is elected, physiologic testing continues to offer valuable supplementary information (Fig. 5–16). If the choice is made to defer operation, a baseline physiologic study should be performed and the patient should be carefully followed to detect disease progression. If an interventional approach, such as arterial reconstruction, balloon angioplasty, laser disobliteration, or sympathectomy, is selected, a preoperative evaluation is of value to establish a baseline against which the therapeutic outcome can be measured. During the operation, at the completion of the reconstruction, physiologic testing may alert the surgeon to the presence of a mechanical defect that might adversely affect the result of the surgery (see Chapter 26).[221] Repeat studies in the recovery room and during the first few days after interventional therapy will permit early recognition of failure. Follow-up monitoring helps identify early failure, deteriorating physiologic parameters that herald impending failure, and the lack of significant improvement despite contin-

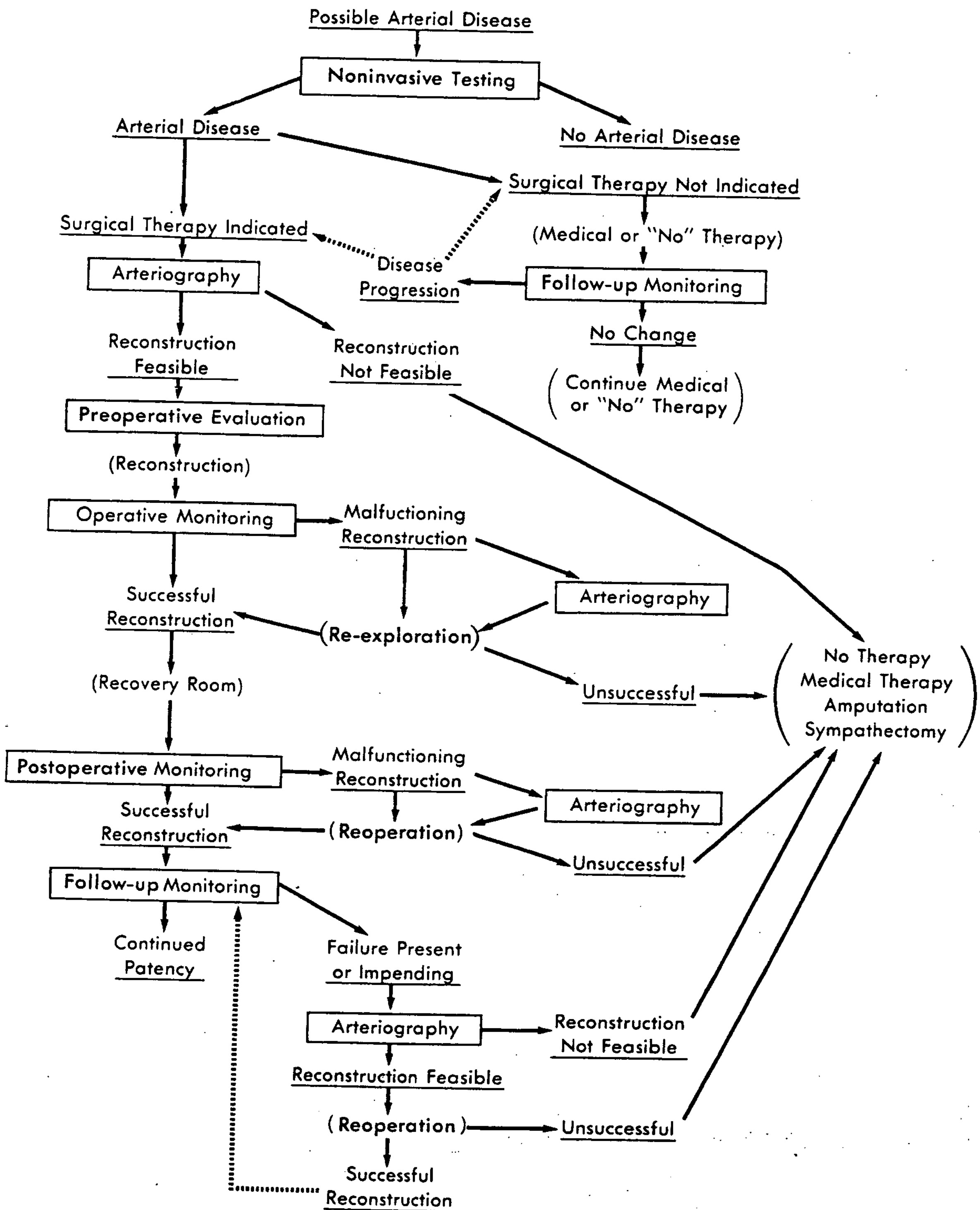


Figure 5–16. Physiologic approach to the diagnosis and management of peripheral arterial disease. In this diagram, diagnostic procedures are enclosed in boxes; conclusions, interpretations, and results are underlined; and therapeutic procedures are enclosed in parentheses. Feedback loops are indicated by broken lines.

ued patency of the reconstruction—any of which may signify the need for arteriographic evaluation and possible repeat surgery.[22, 170, 228] Objective assessment of the physiologic improvement resulting from arterial reconstruction or other interventional measures supplies valuable data that can be used by the surgeon to perfect the therapeutic approach to specific vascular problems.[227, 228]

Although many different methods for studying the arterial circulation have been discussed in this chapter, not all are indicated in the evaluation or follow-up of each patient.[19] Some tests are not as accurate as others, some are more difficult to perform, and many provide overlapping information or information that is not pertinent to the questions being asked. The best policy is to select those modalities that supply the most information, have been shown to be reliable by critical prospective evaluation, and are known to be economical in terms of time and money. The remainder of this chapter is devoted to the application of these tests to specific arterial problems.

Intermittent Claudication

When the presenting complaint is limited to exercise-induced pain or fatigue in the muscles of the leg or buttocks, the two most important questions are: (1) is arterial disease present? and (2) is it severe enough to account for the discomfort? If the ankle pressures are clearly normal, further investigation is seldom required.[172, 222] If, however, the symptoms are highly suggestive of intermittent claudication, or if the ankle pressure is equivocal, the patient should undergo a treadmill exercise evaluation at 2 mph up a 10 per cent grade (Fig. 5–17).[41] A drop in ankle pressure indicates that blood flow is inadequate to sustain normal metabolism during walking, confirming the diagnosis of claudication. If, on the other hand, the resting ankle pressure is clearly abnormal, exercise testing is required only to substantiate the diagnosis, to evaluate the extent of the disability, and to provide a baseline for follow-up studies. When, despite a drop in ankle pressure, the patient stops walking because of angina, dyspnea, or pain in the hip or knee, the limiting cardiac, pulmonary, or orthopedic defect is treated first before arterial reconstruction is considered. Cessation of walking with little or no drop in ankle pressure suggests neurogenic claudication (pseudoclaudication).[44, 84, 123, 230] Rarely, the ankle pressure may not drop even though the resting ankle pressure is decreased. This may occur when the arterial obstruction is limited to the distal vessels of the leg below the origin of the major blood supply to the calf muscles (sural arteries). In this event, the more proximal segmental pressures, plethysmographic tracings, and Doppler velocity signals will usually be normal. These patients may complain of foot claudication. Foot claudication in conjunction with normal ankle pressures is also seen in some patients with thromboangiitis obliterans in whom the obstruction is limited to the pedal arteries.[211]

A more common cause of a "normal" resting ankle pressure in limbs with symptoms suggestive of claudication is medial calcification in patients with diabetes. In such cases, abnormal Doppler signals or abnormal segmental or digital plethysmographic waveforms can be used to substantiate the diagnosis. Cutaneous oxygen tension measurements, however, are often normal at rest, even in limbs with significant proximal arterial disease and definite claudication.[36] A drop in the $tcPo_2$ after treadmill exercise and a delayed recovery are quite specific for intermittent claudication, even in diabetic extremities (see Table 5–17).[36, 99]

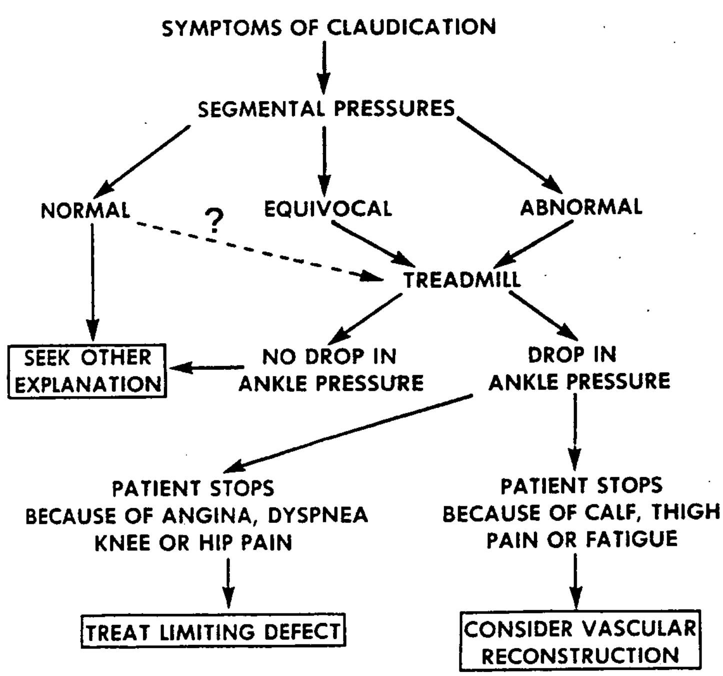


Figure 5–17. Algorithm illustrating diagnostic approach to patients with claudication. (Modified from Sumner DS: Algorithms using non-invasive diagnostic data as a guide to therapy of arterial insufficiency. *In* Puel P, Boccalon H, Enjalbert A (eds): Hemodynamics of the Limbs-1, Toulouse, France, GEPESC, 1979, pp 543–566.)

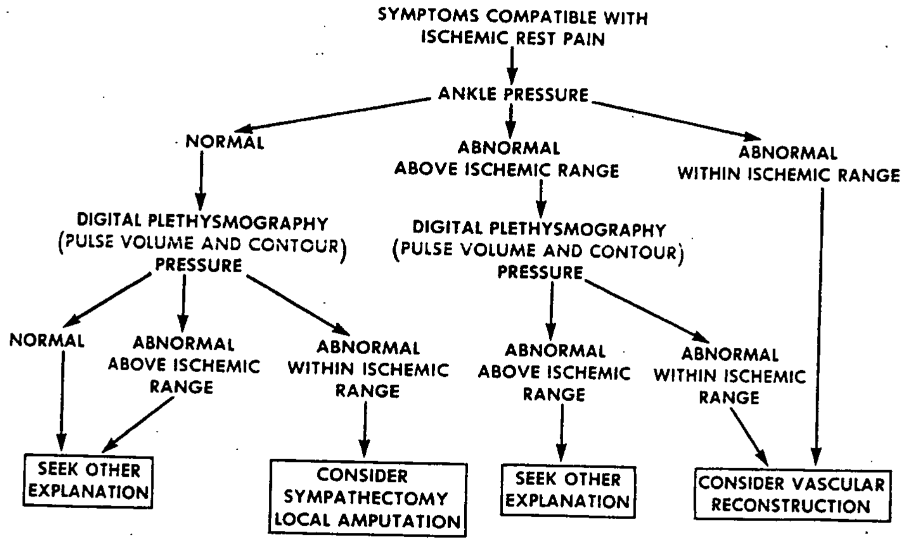


Figure 5–18. Algorithm illustrating diagnostic approach to patients with rest pain of the feet.

Raines and associates concluded that limiting claudication was unlikely if the postexercise ankle pulse volume recordings fell into categories 2 or 3; but that claudication was probable when the recordings fell into categories 4 or 5 (Table 5–14).[180]

As discussed previously in this chapter, reactive hyperemia tests can be substituted for treadmill exercise to evaluate the severity of the obstructive process. Unfortunately, reactive hyperemia testing does not provide information regarding other nonvascular conditions that limit walking and, in my opinion, is rarely indicated. If the patient cannot walk on the treadmill, treatment for claudication is not indicated and the information supplied by reactive hyperemia is superfluous. Nonetheless, the half-time required for recovery to preischemic pulse volumes or $tcPo_2$ values have been shown to discriminate well between normal and claudicating extremities; therefore, these tests may have a role in the evaluation of diabetic patients with incompressible arteries.[47, 75, 134]

It should be noted that the absolute level of the ankle blood pressure is of less importance than the ankle pressure index in assaying the severity of claudication. For example, a patient with a brachial systolic pressure of 100 mmHg and an ankle pressure of 110 mmHg will have no claudication, but a patient with an arm pressure of 150 mmHg and an ankle pressure of 110 mmHg will be significantly limited by claudication. Although both will have normal resting blood flows, the muscle blood flow during exercise in the patient with an ankle index of 0.73 will be restricted (see Chapter 3).

Rest Pain

Absence of pedal pulses, dependent rubor, trophic skin changes, and decreased skin temperature—all hallmarks of severe ischemia—are generally present in patients with rest pain, even when there is no evident tissue loss. When the ischemia has been present for only a short time, the skin may appear relatively normal. Other causes of pain, such as orthopedic problems and infections, are usually easily recognized, but neuropathic pain associated with diabetes mellitus may present a diagnostic challenge, particularly in limbs with concomitant arterial obstruction. The function of noninvasive testing is not only to identify the presence of arterial obstruction but also to ascertain whether the blood flow at rest is sufficiently compromised to be responsible for the symptoms.

Again, the first step is to measure the ankle systolic pressure (Fig. 5–18).[222, 223] Stress testing is not necessary; subtle degrees of arterial obstruction are of no concern. If the ankle pressure is clearly within the ischemic range, vascular reconstruction should be strongly considered. When the ankle pressure is normal or when it is abnormal (below arm pressure) but above ischemic levels, digital plethysmography should be employed and toe pressures obtained. If the toe pressures and plethysmographic tracings are normal or are abnormal but above the ischemic range, it is most unlikely that that patient has an ischemic foot (see Fig. 5–6).[183] In the absence of a skin lesion, other explanations for the pain must be sought. If, on the other hand, toe pressures and plethysmographic tracings are compatible with severe ischemia, arterial reconstruction should be considered in any limb with a decreased ankle pressure, since even a moderate elevation of the perfusion pressure may relieve pain. In limbs with ischemic feet but normal ankle pressures, therapeutic options are limited to amputation or possibly to sympathectomy (in cases of mild rest pain).

Measurements of $tcPo_2$ levels from the skin of the dorsum of the foot may be diagnostic of rest pain and may also provide an objective assessment of the degree of ischemia (see Table 5–16).[36, 47, 253] Such measurements are likely to be of most value in diabetic extremities, in which pressure data are frequently unreliable.[47] In the same situation, the diagnosis of rest pain is probable when the ankle pulse volume recordings are in the range of categories 3 to 4 and is likely when the

categories are in the range of 4 to 5 (see Table 5–14).[180] Laser Doppler assessment may also be of value, but clinical experience with this technique is limited.[118]

There is no hard and fast definition of what constitutes an ischemic ankle pressure. Certainly, pressures less than 35 mmHg in nondiabetic patients and less than 55 mmHg in diabetic patients must be regarded as ischemic or nearly so.[180] The use of the ankle/brachial index is inappropriate, since the critical concern is not the severity of the obstructive process but rather the head of pressure available to pump blood through the pedal arteries and the vascular bed of the foot. A patient with a high systemic blood pressure may have an adequate ankle pressure despite a markedly reduced ankle pressure index.

Ischemic Ulcers and Gangrene

Foot ulcers may be neuropathic, ischemic, or have a combined neuropathic-ischemic etiology. Although purely neuropathic ulcers are usually easily distinguished from those due to ischemia by their characteristic location and the normal appearance of the surrounding skin, many foot ulcers are not so readily categorized. Gangrene, on the other hand, always implies ischemia; but its presence does not define the location or extent of the occlusive process responsible for the impaired blood flow. The role of noninvasive testing, therefore, is to ascertain whether ischemia is present, to determine its severity, and to assess the potential for healing.

The basic approach is similar to that employed in the evaluation of rest pain (Fig. 5–19).[222] Ankle pressures are used to identify obstructions in arteries above the foot, and toe pressures and digital plethysmography are used to determine the degree of ischemia in the feet of limbs with normal ankle pressures or in limbs with abnormal but "not ischemic" ankle pressures.[183] Determination of $tcPo_2$ values or pulse volume recordings may be used instead of, or in conjunction with, the digital studies. When the ankle pressure is ischemic (<40 to 50 mmHg in nondiabetics or <80 mmHg in diabetics) or when the ankle plethysmogram is markedly abnormal or absent (pulse volume categories 4 or 5), there is little hope that ulcers will respond to local treatment or that forefoot amputations will be successful.[180] In this event, arterial reconstruction—if technically feasible, safe, and otherwise not contraindicated—should be performed in order to avoid a major amputation.[165]

Arterial reconstruction should also be considered in limbs with abnormal but not ischemic ankle pressures when the digital pressures, toe plethysmograms, or $tcPo_2$ values are indicative of severe pedal ischemia (Fig. 5–19). Despite the presence of significant pedal arterial disease, the increase in pressure resulting from a successful arterial reconstruction is often sufficient to ensure the success of local therapy. Even when healing can be expected on the basis of pedal hemodynamic parameters, arterial reconstruction may accelerate the healing process.

In limbs with normal ankle pressures or normal ankle plethysmograms, arterial reconstruction will not increase blood flow to the foot. If the toe or $tcPo_2$ studies are normal or, if abnormal, are not indicative of ischemia, there should be no problem with healing. Ischemic values, however, present a different problem. In this situation, ischemia is attributable solely to digital or pedal arterial obstruction; reconstruction at this level is not feasible and a Symes' or below-knee amputation is usually required. Sympathectomy is seldom if ever beneficial.

Criteria for Predicting Healing of Foot Lesions or Forefoot Amputations

A blood supply adequate to ensure healing under ideal circumstances may be insufficient when complicating factors, such as infection, diabetes, poor nutrition, or

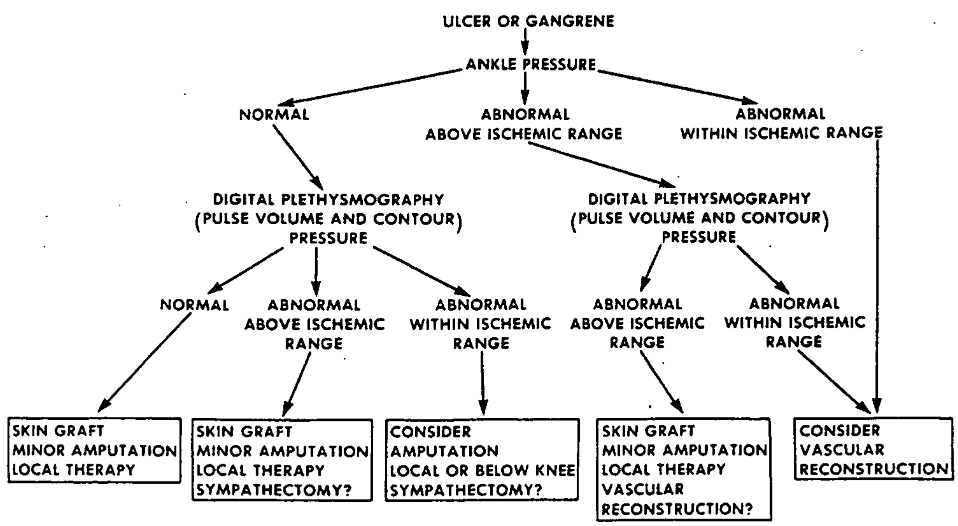


Figure 5–19. Algorithm illustrating diagnostic approach to patients with ulcers or gangrene of the feet. (From Sumner DS: Algorithms using non-invasive diagnostic data as a guide to therapy of arterial insufficiency. *In* Puel P, Boccalon H, Enjalbert A (eds): Hemodynamics of the Limbs-1, Toulouse, France, GEPESC, 1979, pp 543–566.)

Table 5–20. Predicting Healing of Foot Lesions and Forefoot Amputations in Diabetic and Nondiabetic Patients

Modality	Author	Criterion†	Correctly Predicted (%) (Sensitivity) Healing	Correctly Predicted (%) (Specificity) Nonhealing	Normalized Predictive Value (%)‡ (Positive) Healing	Normalized Predictive Value (%)‡ (Negative) Nonhealing
Ankle pressure	Cumulative[a]	80 mmHg	79	63	80	62
	Cumulative[b]	70 mmHg	68	40	68	40
	Cumulative[c]	60 mmHg	87	46	75	66
Toe pressure	Cumulative[d]	40 mmHg	97	81	91	94
	Cumulative[e]	30 mmHg	73	87	91	64
	Schwartz (1982)[199]	20 mmHg	96	80	90	92
$tcPO_2$	Karanfilian (1986)[119]	10 mmHg	100	88	94	100
	Harward (1985)[96]	10 mmHg	71	57	75	51
Skin pressure	Faris (1985)[67]	40 mmHg	97	80	90	94
Elevation	Gilfillan (1985)[83]	35 cm	96	85	92	92
Laser Doppler	Karanfilian (1986)[119]	40 mv	79	96	97	71
PVR	Gibbons (1979)[82]	Moderate	46	94	93	48

*References: a, 28, 67, 106, 183, 199, 242; b, 82, 159; c, 9, 17, 42, 83; d, 28, 67; e, 17, 42, 106, 183.
†Level above which healing is expected.
‡Assumes 65% healing rate.

local trauma, are present. Owing to localized end-artery obstruction, some areas of the foot may be ischemic while others remain adequately perfused. For example, a heel ulcer can exist and be recalcitrant to treatment in a foot with viable toes. The skill of the surgeon and compliance of the patient are also immensely important. It is not surprising, therefore, that noninvasive criteria for predicting healing vary from laboratory to laboratory. In view of the multiple factors determining healing potential, this should not be considered an indictment of the tests; rather, it emphasizes that the results of noninvasive tests should be used as guidelines—nothing more—in predicting the outcome of foot lesions and amputations. Nevertheless, when used in conjunction with clinical observations, noninvasive test results are frequently quite helpful to the surgeon who is faced with the perplexing decision of whether to revascularize the extremity, amputate immediately, or persist with nonoperative therapy.

Table 5–20 lists criteria that have been used by various investigators to discriminate between lesions or amputations that are likely to heal and those that are not. Predictive values obtained from the raw data are normalized to a 65 per cent healing rate and a 35 per cent failure rate. These figures represent the average distribution of healing and failure based on clinical assessment alone, as reported by the investigators whose data are included in the table. (The healing rate varied from 35 to 84 per cent.)

As indicated in Table 5–20, ankle pressures tend to be less predictive than tests that are designed to sense perfusion of the foot itself. This is particularly true in diabetic limbs for two reasons: the arteries at ankle level are often incompressible, giving spuriously high values, and pedal arterial obstruction, which is frequently present, may be responsible for severe ischemia even in the absence of disease above the ankle. Moreover, there is little or no correlation between ankle pressures and pedal skin blood flow.[246] Even when the ankle pressure exceeds 80 mmHg, a failure rate of about 20 per cent can be expected. While ankle pressures less than 60 to 80 mmHg accurately predict failure in only about 60 per cent of cases, the potential for healing is markedly reduced at lower levels. For example, Carter reported that all limbs with ischemic lesions required amputation when the ankle pressure was less than 55 mmHg, and Holstein and coworkers found that only 11 per cent of skin lesions healed when the pressure fell below 50 mmHg.[42,106] It is very unlikely that healing will occur at foot level in any limb with an ankle pressure less than 30 or 40 mmHg (see Fig. 5–7).[183,242]

With tests of more peripheral vessels, it is generally possible to define a limit above which healing is to be expected, but the converse is somewhat less likely to be true. In other words, healing may occur when the noninvasive parameters are below the "critical" level (see Table 5–20). There are at least three possible explanations for these observations: (1) the test results are inaccurate, (2) the test does not measure the proper parameter, and (3) the minimum value required for healing may be very low under ideal circumstances. For these reasons, most surgeons are reluctant—and rightly so—to base their decision to proceed to a major amputation exclusively on noninvasive test findings.

Raines and associates suggest that healing of foot lesions is likely with PVR categories 1 and 2, probable with category 3, and unlikely with categories 4 and 5 when tracings are obtained at ankle level (see Table 5–14).[160] While strong or moderate forefoot pulse volumes were found to be highly predictive of successful healing by Gibbons and associates, absent or low-volume waveforms predicted failure in only 48 per cent of the diabetic limbs included in their study (see Table 5–20).[82] In our experience, toe plethysmographic tracings are equally predictive (see Fig. 5–15).[223]

Carter found that foot lesions usually heal if toe pressures exceed 30 mmHg in nondiabetic patients or

55 mmHg in diabetics.[42] On the other hand, Holstein and associates noted no appreciable difference between the two groups.[106] In their study, healing occurred in 91 per cent of the limbs when toe pressures were greater than 30 mmHg, in 50 per cent when pressures were between 20 and 29 mmHg, and in only 29 per cent when pressures were less than 20 mmHg. Bone and Pomajzl noted failure of toe amputations in all cases in which the toe pressures were less than 45 mmHg and in 25 per cent of cases with pressures between 45 and 55 mmHg; healing occurred in all cases with toe pressures greater than 55 mmHg.[28] Other investigators have reported uniform healing when toe pressures exceeded 10 to 25 mmHg.[17, 199] Several successful toe amputations have been reported in toes with undetectable pressures.[17] Since the pressure in any gangrenous digit will almost certainly be zero, it is wise to use the pressure from nongangrenous adjacent digits to predict healing.[28, 199]

In our laboratory, toe pressures have proved to be of more prognostic value than ankle pressures (see Fig. 5–7).[183] Lesions and toe amputations failed to heal in 92 per cent of limbs with an ankle pressure less than 80 mmHg, but they also failed to heal in 45 per cent of limbs with higher pressures. There were three failures in limbs with ankle pressures of 150 mmHg; in all three cases, the toe pressures were less than 30 mmHg. When toe pressures were less than 30 mmHg, the failure rate was 95 per cent, but when the pressures were above 30 mmHg, only 14 per cent of the lesions or amputations did not heal. Our experience suggests that toe pressures below 20 mmHg almost uniformly predict an unsuccessful result.

While transcutaneous oxygen tension measured at foot level is a promising method for ascertaining healing potential, the minimal value consistent with a successful result remains uncertain. For example, the discrimination provided by a $tcPo_2$ of 10 mmHg was excellent in the study by Karanfilian and coworkers,[119] but was less so in the study by Harward and associates (see Table 5–20).[96] Other investigators have suggested that healing is unlikely if the $tcPo_2$ is less than 20, 40, or 50 mmHg.[47, 73, 121] Hauser reported uniform failure of amputations when the RPI was less than 0.40 and nearly complete success when the index exceeded 0.60.[97] Laser Doppler, another technique for assessing skin blood flow, was found by Karanfilian and coworkers to be somewhat less accurate than $tcPo_2$ in a comparative study of the two modalities.[119]

An interesting and apparently highly predictive method of measuring foot perfusion pressure has been advocated by Gilfillan and associates (see Table 5–20).[83] With the patient supine, the foot is elevated 65 cm above the level of the right atrium, kept in this position until the foot blanches, and then lowered gradually until the color returns. At the point at which color returns, the perfusion pressure in centimeters of blood is equivalent to the height of the foot above the atrium. Listening for the return of blood flow in the pedal arteries with the Doppler flowmeter is a variation of this technique that we have occasionally employed in diabetic limbs with incompressible vessels. Isotope clearance methods for measuring skin blood pressure, as reported by Faris and Duncan, have also proved efficacious (see Table 5–20).[67] These techniques are further discussed in Chapters 8 and 149.

Since rigid guidelines cannot be established, it is best to think of the results of the various noninvasive tests as being indicative of the probability that a foot lesion or forefoot amputation will heal. When the probability is low, vascular reconstruction should be performed if at all possible (see Fig. 5–19). Even a small increase in perfusion may tip the balance in favor of healing. Noer and associates, for example, found that foot ulcers gradually healed when, as a result of vascular reconstruction, the toe pressure rose 15 to 20 per cent of the arm pressure, an increase of only 20 to 30 mmHg.[165]

Criteria for Predicting Healing of Major Amputations

Major amputations become necessary in limbs with severe ischemia or gangrene when arterial reconstruction is not feasible or when the extent of tissue destruction precludes salvage by localized amputation. Since the chance of rehabilitation is better the more distal the amputation site, Symes' amputations are preferred over below-knee, and below-knee over above-knee amputations. Unfortunately, the potential for healing is just the opposite, diminishing as the site of amputation moves from proximal to distal levels of the leg. Keagy and coworkers, reporting on a large series from a major university hospital, found that the failure rate for above-knee amputations (AKA) was 9 per cent compared with 19 per cent for below-knee amputations (BKA).[124] These findings are in basic agreement with those of other investigators, who, like Keagy and his colleagues, selected the amputation site exclusively on the basis of clinical judgment. Although many factors determine the success or failure of an amputation, it is obvious that healing will not occur without an adequate blood supply. Various noninvasive methods of assessing perfusion at the intended site of amputation have, therefore, been proposed in an effort to decrease the number of failures.

Almost all limbs with a Doppler-derived calf systolic pressure in excess of 65 to 70 mmHg will heal a below-knee amputation.[16, 17, 82, 159, 180] It is, however, difficult to define a calf pressure below which BKAs consistently fail. Although Raines and associates[180] have stated that healing is unlikely when the calf pressure is less than 65 mmHg, Gibbons and associates[82] noted no failures in 11 limbs with pressures less than this. Nicholas and colleagues found that 65 per cent of the BKAs with pressures less than 70 mmHg in their series healed.[159] Barnes and coworkers reported a 75 per cent healing rate when the below-knee pressure was less than 70 mmHg and a 44 per cent healing rate when the pressure was less than 50 mmHg.[16] In a subsequent study, Barnes and coworkers found no significant difference between the mean below-knee pressures of those limbs that healed a BKA and those that did not.[17] In fact, eight limbs with no

recordable calf pressure healed. This, of course, does not mean that there was no blood pressure at the site of the amputations; it does imply that the velocity of flow below the cuff was insufficient to obtain a Doppler signal.

As one would expect, a low (or zero) ankle pressure does not necessarily predict an unsuccessful BKA, since the pressure at the more proximal amputation site may be entirely satisfactory.[17, 62, 82, 159, 177] On the other hand, a reliable ankle pressure (i.e., one that is not artificially elevated because of arterial calcification) that exceeds 30 mmHg is a favorable prognostic sign, because the pressure at all proximal levels will exceed this value.[159, 180]

If the data obtained from skin blood pressure measurements can be assumed to be representative of the actual perfusion pressure, the circulatory support necessary to heal below-knee amputations is quite low indeed. For example, Holstein and coworkers found that healing of below-knee amputations occurred in 25 per cent of limbs with skin pressures less than 20 mmHg, in 67 per cent with pressures between 20 and 30 mmHg, and in 90 per cent with pressures greater than 30 mmHg.[105] They used the clearance rate of a radionuclide injected intradermally 10 cm below the knee joint to monitor the cessation of cutaneous blood flow as pressure on the skin was increased with a pneumatic cuff (see Chapter 8). Similar results were reported by Stockel and associates, who used a photodetector to detect the return of blood flow as the counter pressure was reduced.[208] Because the critical below-knee pressure seems to be less than 20 mmHg, it may be impossible to measure it accurately with the usual noninvasive methods.

Raines and coworkers have stated that healing of below-knee amputations is unlikely if the pulse volume tracing obtained at calf level is flat or shows a minimal pulse and the below-knee pressure is less than 65 mmHg.[180] Others, however, have found pulse volume recordings to have little predictive value.[82, 159]

Transcutaneous oxygen tension measurements are plagued with much the same problems that beset pressure measurements. Although most investigators agree that over 90 per cent of BKAs will heal when the $tcPo_2$ obtained from the skin of the calf about 10 cm below the knee is greater than 35 to 40 mmHg, a minimal level below which failure is to be expected has proved more difficult to define.[47, 97, 121, 156, 184] The level most often cited is 30 to 35 mmHg. However, Ratliff and associates[184] observed that 67 per cent of the BKAs in limbs with $tcPo_2$ values less than 35 mmHg healed, and Harward and coworkers[96] reported that 82 per cent with values less than 10 mmHg healed. In fact, Harward's group found that 98 per cent of the BKAs with $tcPo_2$ values greater than 10 mmHg were successful. Of those that healed, 55 per cent had midcalf $tcPo_2$ values below 30 mmHg and 4 per cent had values of zero, even after oxygen inhalation. Malone and associates, in a recent study of above-knee, below-knee, and transmetatarsal amputations, found that $tcPo_2$ values of 20 mmHg provided perfect separation between those limbs that healed and those that did not.[143] The clearance rate of xenon-133, a technique previously advocated by their group, was not reliable as a prospective test for selecting the level of amputation (see Chapters 8 and 149).

In summary, a calf pressure greater than 40 mmHg, an ankle pressure greater than 30 mmHg, or a midcalf $tcPo_2$ greater than 35 mmHg provides reasonable assurance that a below-knee amputation will heal; however, lower values should not deter the surgeon from attempting an amputation at this level if other signs are favorable.

Acute Arterial Obstructions

The clinical diagnosis of acute arterial obstruction is seldom difficult and is usually easily made on the basis of the history and physical examination. Simple noninvasive tests, however, are frequently useful in an adjunctive role.

Embolism and Thrombosis

It is sometimes difficult to distinguish between an embolic obstruction and one caused by thrombosis of a previously stenotic artery. Finding normal pulses, Doppler signals, and ankle pressures in the opposite extremity suggests that the process is likely to be embolic. Abnormal findings in the opposite extremity, indicating the presence of chronic occlusive arterial disease (which is often bilateral), suggests thrombosis. This distinction is important since the therapeutic approaches to the two conditions differ. Although preoperative arteriography is seldom necessary if the obstruction is embolic, angiography is very helpful in cases of thrombotic obstruction, which often require arterial reconstruction.

A quick survey of the involved limb with the Doppler flowmeter will usually identify the site of obstruction. This information can influence the choice of incision. Although a palpable pulse may be present (for example, in the femoral artery), no flow may be detected, indicating that the underlying artery is occluded. An abrupt, "thumping" signal strongly suggests that all or nearly all of the outflow is occluded. This finding is typical in cases of embolic obstruction of the distal common femoral artery in which both the superficial femoral and profunda femoris arteries are occluded.

Although unrelenting pain, loss of sensation, and reduced motor function indicate severe ischemia and demand immediate surgical intervention, there are situations in which ischemia is present but is not immediately limb threatening.[214] In these cases, there may be some subjective numbness and coldness of the foot but no objective loss of sensation, limitation of movement, or pain. When velocity signals are present in the pedal arteries and when the ankle pressure exceeds 30 or 40 mmHg, it is possible to temporize, since collateral circulation is adequate to sustain viability—at least for the moment. The time gained may be used to improve the patient's condition. Such pa-

tients may also be candidates for thrombolytic therapy. An ankle pressure index greater than 0.50 indicates excellent collateral circulation.[214] Flow will improve even if nothing is done (although the patient may subsequently experience claudication). If, on the other hand, no velocity signals are obtained from the pedal arteries or if the systolic pressure is less than 30 mmHg, implying marginal collateral input, the surgeon should move expeditiously to restore circulation.

Occasionally, patients with a low cardiac output present with symptoms and signs suggestive of an acute occlusion when in fact there has been no anatomic change in the status of the limb circulation. In this situation, there is almost invariably a substrate of pre-existing chronic arterial disease. Although one leg may be worse than the other, both are usually involved. Doppler signals are usually present in the pedal arteries and a Doppler survey of the common femoral, superficial femoral, and popliteal arteries will reveal no site of acute occlusion, although the signals in these areas are often abnormal. Because of pre-existing arterial disease, the ankle pressure index is usually low and the absolute ankle systolic pressure may fall in the ischemic range. With correction of the cardiac failure, the ankle pressure will improve even though the index does not change. Diagnosis in these patients can be difficult, and frequent careful monitoring during the period of resuscitative therapy is required to avoid missing an acute occlusion.

Trauma

Finding reduced or absent velocity signals in arteries distal to the site of penetrating or blunt limb trauma indicates the need for arteriography or surgical exploration. (The mere presence of a Doppler signal never rules out an arterial injury.) Even when peripheral pulses are palpable, a decreased systolic blood pressure in the limb below the area of injury is also highly suggestive of arterial damage. It must be remembered, however, that the Doppler survey is useful mainly when it is positive; a negative study does not exclude arterial trauma when there is major hemorrhage, hematoma formation, penetrating wounds or fractures in the vicinity of the arterial supply, or other indications for surgical exploration.[14]

In patients in whom only one of the three below-knee arteries has been injured, noninvasive tests can be helpful in evaluating the adequacy of the residual perfusion to the foot; a normal or near normal study provides reassurance to the surgeon, who may elect to ligate rather than reconstruct the injured vessel. A Doppler study can also identify which of the vessels are damaged. It must be emphasized that the presence of Doppler signals does not eliminate the possibility of a developing compartment syndrome.[14]

During the Vietnam conflict, the ultrasonic flow detector proved to be a valuable method for ascertaining the viability of limbs in which palpable pulses did not return or were lost after arterial repair. In the experience reported by Lavenson and associates, all extremities with audible signals remained viable, whereas all extremities with absent signals required further reconstruction or amputation.[138] Thus, noninvasive examination identified those limbs in which it was safe to defer definitive arterial repair long enough to permit control of infection.

Kram and Shoemaker have advocated the use of transcutaneous oxygen tension measurements as an adjunctive diagnostic method in patients with trauma of the limbs.[133] They found that the mean $tcPo_2$ in limbs with arterial injury (34 ± 21 mmHg) was significantly lower than that in traumatized limbs without arterial injury (58 ± 14 mmHg). A ratio of 0.90, obtained by dividing the $tcPo_2$ in the injured limb by that in the opposite uninjured extremity, identified major arterial injury with a sensitivity and specificity of 80 per cent and 91 per cent, respectively. It would seem, however, that $tcPo_2$ measurements would be more appropriately used to assess the viability of a traumatized limb rather than to detect arterial injury.

Diagnostic Arterial Catheterization

Ankle pressures and brief Doppler surveys help identify unsuspected arterial obstruction that could make catheterization difficult or hazardous, in which case, another limb can be used.[15] After the procedure, pressure data can be used to determine whether an accident has occurred and to help formulate a therapeutic approach. With spasm, the blood pressure drops only moderately and recovers rapidly. When peripheral pressures are above 40 mmHg, one can afford to wait an hour or two to see whether clot lysis will take place. If there is no response or if the initial pressures are quite low, immediate repair is indicated. When the pressure index is greater than 0.50, surgical intervention is not required for limb preservation and can be delayed if there are significant medical contraindications.

Not infrequently, it is difficult to distinguish between a hematoma at the groin and an early false aneurysm. In these cases, duplex scanning (particularly with instruments that color-code the flow) has proved to be extremely valuable. Although a hematoma may appear to pulsate and may have a configuration similar to that of a false aneurysm, no flow will be detected; flow within the mass identifies it as a false aneurysm.

Popliteal Entrapment and Adventitial Cystic Disease

Popliteal artery entrapment should be considered when young men present with intermittent claudication (see Chapter 66). The claudication may be atypical, occurring with walking but not with running; and peripheral pulses may be normal.[149] In these cases, noninvasive tests can be used to demonstrate compression of the popliteal artery by the medial head of the gastrocnemius muscle. Hyperextension of the knee, passive dorsiflexion of the ankle, and active plantar flexion of the foot with the knee moderately flexed are three maneuvers that have little effect on the distal circula-

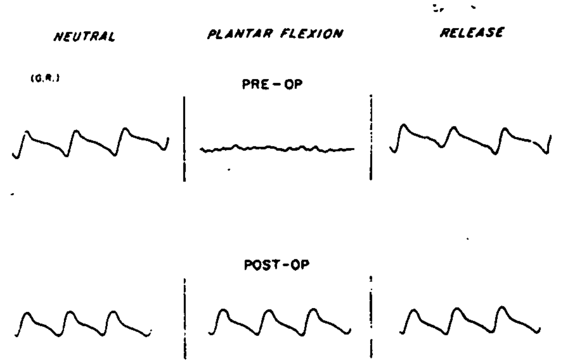


Figure 5–20. Pulse volume recordings at ankle level before and after surgery for popliteal artery entrapment. Prior to operation, pulses disappear during plantar flexion. After surgical correction, plantar flexion has no effect on pulse volume.

tion of normal limbs but decrease the ankle systolic blood pressure, diminish the amplitude of the pedal arterial Doppler signals, and reduce the volume of pulses recorded at the ankle in limbs with popliteal entrapment (Fig. 5–20).[50, 59, 151] Unfortunately, these maneuvers, if conducted very vigorously, may produce positive results even in normal extremities, leading some investigators to question their reliability.[149] As an alternative, exercise testing has been recommended. A drop in the ankle pressure index after treadmill exercise at 3 miles per hour at a zero grade or at 4 miles per hour at a 10° grade documents the presence of intermittent or fixed arterial obstruction.[149] A negative response, on the other hand, provides reasonable assurance that the patient does not have the popliteal entrapment syndrome.

Cystic adventitial disease of the popliteal artery is another cause of intermittent claudication that characteristically affects young athletic males (see Chapter 65). Unlike popliteal entrapment, circulatory changes are typically absent when the leg is extended but develop when the knee is sharply flexed.[20] Segmental pressures, Doppler-derived waveforms, and plethysmographic pulses are normal above the knee and abnormal below. Treadmill exercise testing will establish the presence of partial arterial obstruction.

Preoperative Evaluation

Once the decision to operate has been made based on the history, physical examination, and noninvasive findings, arteriography is the next logical step. An anatomic assessment of the distribution of disease is necessary to determine the feasibility of reconstruction and to plan the operative approach. Unfortunately, the preoperative arteriogram may provide insufficient information in two commonly encountered stiuations: (1) severe peripheral ischemia with below-knee obstructive disease, and (2) multilevel disease involving both the aortofemoral and femoropopliteal segments.

Identification of Recipient Arteries for Femoral-Distal Bypass

Despite the best efforts of the angiographer to enhance blood flow pharmacologically or by reactive hyperemia, arteriographic visualization of the distal crural vessels may be inadequate in limbs with gangrene, ischemic ulcers, or rest pain. Ricco and coworkers reported complete nonvisualization of calf vessels in 20 per cent of preoperative arteriograms and limited visualization in another 20 per cent.[187] In the majority of such limbs, a velocity signal can be obtained with the Doppler probe from one or more of the pedal arteries, indicating the presence of a residual channel that is at least partially patent. The presence of a signal does not, however, always identify a vessel suitable for accepting a graft. The lumen may be too small or the communications with the remainder of the pedal vessels too poor to ensure patency of the graft or to provide adequate perfusion for the foot. Occasionally, the signal may arise from a neighboring collateral artery. Nonetheless, the correlation between the results of carefully conducted Doppler surveys and the findings obtained with intraoperative arteriography or surgical exploration are gratifyingly high.[187, 201] In the series reported by Ricco and associates, Doppler examination correctly identified arteries suitable for bypass in 71 per cent of the limbs with nonvisualization of distal vessels and in 83 per cent of the limbs in which preoperative angiographic visualization was inadequate (Fig. 5–21).[187] A simple scoring system developed by Shearman and colleagues, based on a combination of clinical data, Doppler signals from the pedal arteries, and the ankle pressure index, was found in a prospective study to be highly predictive of the need for a bypass to the popliteal trunk, to the popliteal trifurcation, or to a distal calf artery.[201] Encouraged by these results, they have curtailed their use of preoperative arteriography in patients requiring distal bypass grafts and have relied instead on intraoperative arteriography.

Doppler surveys are initiated by examining the dorsalis pedis and posterior tibial arteries at ankle level. To get some idea of the length of vessel available for bypass and to exclude the possibility that the signal is arising from a collateral channel, one can attempt to follow the signal up the leg or down into the foot. It is often possible to trace the medial plantar artery for a short distance beyond its origin from the posterior tibial and to follow the dorsalis pedis distally in the first metatarsal space to the origin of the deep plantar artery. The patency of the peroneal artery, which cannot be examined directly, can be determined by placing the probe over the anterior lateral malleolar artery. Although the ankle pressure obtained from these arteries may provide some insight into the status of their inflow, medial calcification often renders this assessment unreliable. If manual compression of the anterior tibial or posterior tibial arteries in the proximal leg just below the popliteal trifurcation has little or no effect on the signal obtained from the dorsalis pedis or posterior tibial arteries at ankle level, it can be assumed that the proximal portion of the artery being compressed is occluded or severely stenotic.[188] On the other hand, augmentation of the velocity signal in one artery during proximal compression of the other indicates that the proximal portion of the artery being evaluated is patent. Not infrequently, the signal from the dorsalis

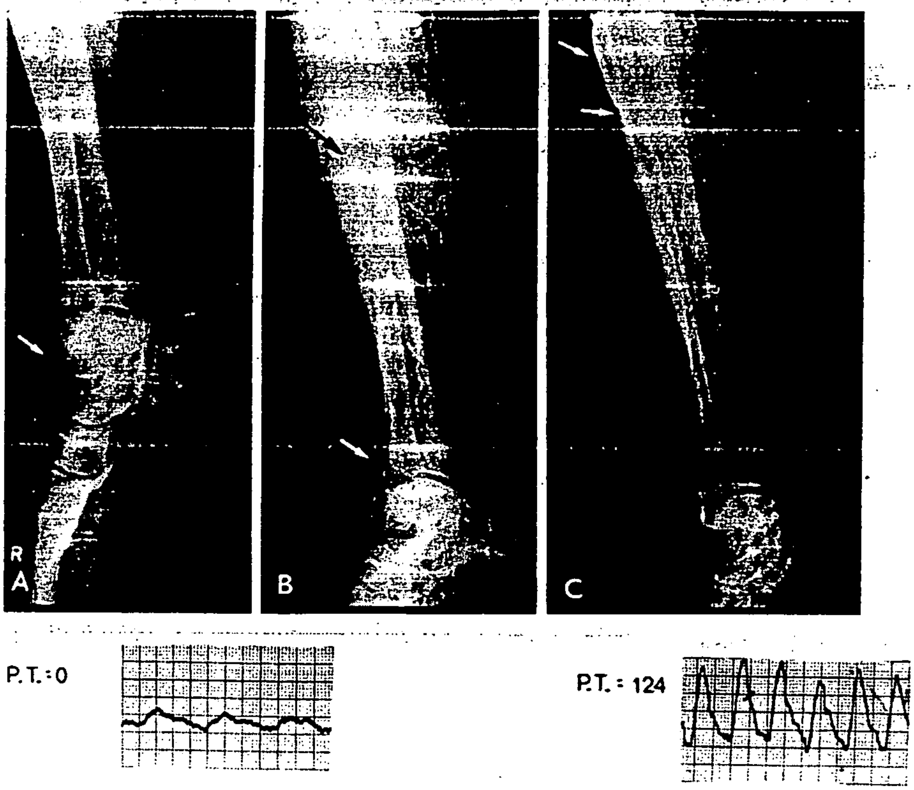


Figure 5–21. Doppler flow detection in femorodistal bypass. *A,* Although the tibial vessels did not fill on arteriography, the Doppler instrument detected pulsatile flow over the dorsalis pedis artery *(arrow)*. *B,* The intraoperative, prebypass arteriogram confirmed the presence of a patent anterior tibial artery *(arrows)*. *C,* Note the change in pulsatile flow patterns after a successful femoral-anterior tibial bypass *(arrows)*.

pedis artery will disappear when the posterior tibial artery is compressed at ankle level (or vice versa), establishing the fact that the compressed artery is serving as the source of inflow for both vessels. In such cases, reversed flow may be detected in one of the two arteries.[188]

Patency of the pedal arch has an important influence on graft function and graft survival. Patency of the pedal arch signifies a low resistance vascular bed and usually implies that the potential for revascularizing the distal foot is good. Yet, even when the preoperative arteriogram opacifies the arteries at ankle level, the pedal arch is seldom adequately visualized. A technique, analogous to Allen's test in the hand, has been proposed by Roedersheimer and associates for assessing the patency of the pedal arch noninvasively.[190] With the Doppler probe applied to the first metatarsal space over the deep plantar artery, the tibial arteries are alternately compressed. If the pedal arch is patent, compression of one of the vessels may attenuate but will not abolish the signal. Disappearance of the signal indicates an incomplete arch supplied by the artery being compressed. They observed a 96 per cent correlation between the results of this test and findings obtained with intraoperative arteriography.

In our laboratory, we have found the color-coded, real-time flow velocity display incorporated in some duplex scanners to be a rapid and accurate method for determining the patency of distal arteries in limbs with severe below-knee disease. A major advantage of this technique is that it also supplies information regarding the diameter of the lumen of the vessels (see Chapter 7).

Multilevel Disease

Failure to recognize a significant inflow lesion adversely affects the chances for patency of a femoropopliteal or femorotibial bypass graft. On the other hand, aortoiliac reconstruction in limbs with hemodynamically significant lesions of the femoropopliteal segment may fail to alleviate symptoms. In 9 to 57 per cent of patients with multilevel disease, reconstructions limited to lesions above the inguinal ligament do not relieve claudication or rest pain and may not allow ischemic ulcers to heal.[40, 64, 79, 108, 130, 131, 192, 198, 227]

Difficulties encountered in interpreting arteriograms of the aortoiliac segment are directly or indirectly responsible for many of the unsatisfactory results.[152] While the surgeon, swayed by the presence of large atheromatous plaques, may overestimate the extent of the disease, it is more likely that he or she will underestimate the degree of iliac arterial stenosis. Asymmetric plaques, which typically lie on the posterior wall, are often not perceptible on the routine anteroposterior projection, their severity becoming apparent only on lateral or oblique views.[32, 200, 238] Although the interpretation of arteriograms of the femoropopliteal segment is more straightforward, the degree of physiologic impairment is greatly influenced by collateral development, which cannot always be reliably estimated. Likewise, palpation of femoral pulses and auscultation of bruits are apt to be unreliable, especially when the examiner is inexperienced.[10, 33, 111, 157, 206]

These problems have spawned a great body of literature concerned with the development of nonin-

vasive methods for ascertaining the relative physiologic significance of concomitant disease in the aortoiliac and femoropopliteal segments. Although it would appear that noninvasive tests are ideally suited for this type of investigation, the results have not been particularly encouraging. In fact, Campbell and coworkers concluded that conventional clinical assessment was more accurate than most noninvasive methods for detecting severe aortoiliac disease; however, laboratory tests were more helpful for lesser degrees of stenosis.[38]

Measurement of ankle pressure immediately following completion of an inflow procedure has been recommended as a method for identifying those limbs that require additional distal bypass.[79] Unfortunately, this simple test has not proved to be as discriminating as originally hoped. Although an immediate rise of 0.10 in the ankle pressure index has consistently been a favorable prognostic sign, no change or even a fall does not necessarily portend a poor result.[4, 79, 131, 227] In fact, an index that is initially decreased may rise to preoperative levels within 1 or 2 hours and may exceed preoperative values within 3 to 24 hours.[131, 166] If, however, the ankle pressure index has not risen by 0.10 in 5 to 7 days, the results of aortofemoral reconstruction are likely to be unsatisfactory.[198] In our experience, the ankle pressure index in 56 per cent of limbs with multilevel disease attained maximal values within 10 days following aortofemoral reconstruction; in another 22 per cent, maximal values were reached in 3 months; and in the remainder, the levels continued to increase for 3 to 26 months.[227]

Theoretically, patients with low upper thigh pressures should have more severe disease in the iliac segment and should benefit more from an inflow procedure than those with high upper thigh pressures. Bone and associates reported that all limbs with an upper thigh pressure index of 0.85 or less were improved after aortofemoral bypass.[29] All unimproved limbs had preoperative thigh indices greater than 0.85, but 65 per cent of those limbs within this range also improved. Others have noted no appreciable difference between the thigh pressure indices in limbs with multilevel disease that improved following aortofemoral reconstruction and those that did not.[167, 227]

It seems logical that poor results would be expected to accompany a large pressure gradient between the upper thigh and the ankle, since this situation implies a high resistance in the arterial segments that lie distal to the aortofemoral reconstruction. In the study reported by Bone and coworkers, 24 per cent of the limbs with one abnormal pressure gradient (>30 mmHg across the thigh, knee, or below the knee) failed to improve.[29] When there were two abnormal gradients, 71 per cent showed no improvement. All proximal reconstructions were successful in limbs with isolated aortoiliac disease (no abnormal gradients). In our study, however, the pressure gradient proved to be of no prognostic value in limbs with multilevel disease.[227]

The fault lies not with the theory but with the inaccuracies inherent in the noninvasive measurement of thigh pressure. When the femoral artery pressure is measured accurately by means of direct arterial puncture and a pressure transducer, the rise in ankle pressure (or toe pressure) following a successful aortofemoral reconstruction is directly proportional to the rise in femoral arterial pressure.[164, 247] Therefore, preoperative knowledge of the arm to femoral arterial pressure gradient and the noninvasively determined ankle pressure can be used to predict the likelihood of a successful result after proximal reconstruction in limbs with multilevel disease.

Because of the fallibility of noninvasive pressure measurements, attention shifted to the interpretation of Doppler-derived common femoral velocity waveforms as a method for determining the hemodynamic significance of lesions within the aortofemoral segment. Simple inspection of the pulse contour is the most direct method (see Fig. 5–9).[68] Persson and colleagues reported that the presence of a triphasic flow pattern with reversed flow in early diastole correctly identified 94 per cent of iliac arteries with either no disease or with stenoses that reduced the diameter by less than 50 per cent.[175] Absence of reversed flow detected stenoses greater than 50 per cent with a sensitivity of 96 per cent. Reverse flow may be absent, however, in limbs with concomitant superficial femoral arterial obstruction despite a normal aortoiliac segment; but in these cases, the systolic portion of the wave is usually normal, displaying a rapid rise, sharp peak, and rapid fall to baseline, and the diastolic portion is usually flat and is not elevated above the "zero" baseline. A triphasic waveform, on the other hand, does not reliably exclude low-grade iliac stenoses of the type that permit normal flow during resting conditions but restrict flow during exercise.[32, 68, 93]

As discussed earlier in this chapter, more sophisticated methods for analyzing the common femoral waveform have been developed. These include pulsatility indices, Laplace transform methods, and principal component analysis. Their accuracies for detecting hemodynamically significant stenoses of the aortoiliac segment are summarized in Tables 5–9, 5–10, and 5–12. Although sensitivities and specificities are generally acceptable, the reported results are quite variable, and it is by no means certain that they are significantly more reliable than simple inspection of the pulse contour. All have been reported to be affected by the presence of superficial femoral arterial occlusions. Thiele and associates found that 92 per cent of limbs with normal femoral pulsatility indices (PI $\geq$ 4) had hemodynamically normal aortoiliac segments as determined by direct measurement of femoral artery pressure after papaverine injection.[231] The presence or absence of concomitant superficial femoral artery occlusion made little difference. Although 88 per cent of limbs with abnormal femoral pulsatility indices (PI < 4) and patent superficial femoral arteries had hemodynamically significant aortoiliac disease, when the superficial femoral artery was occluded, only 45 per cent of the limbs with abnormal pulsatility indices were found to have significant inflow disease. From these data they devised an algorithm that should afford

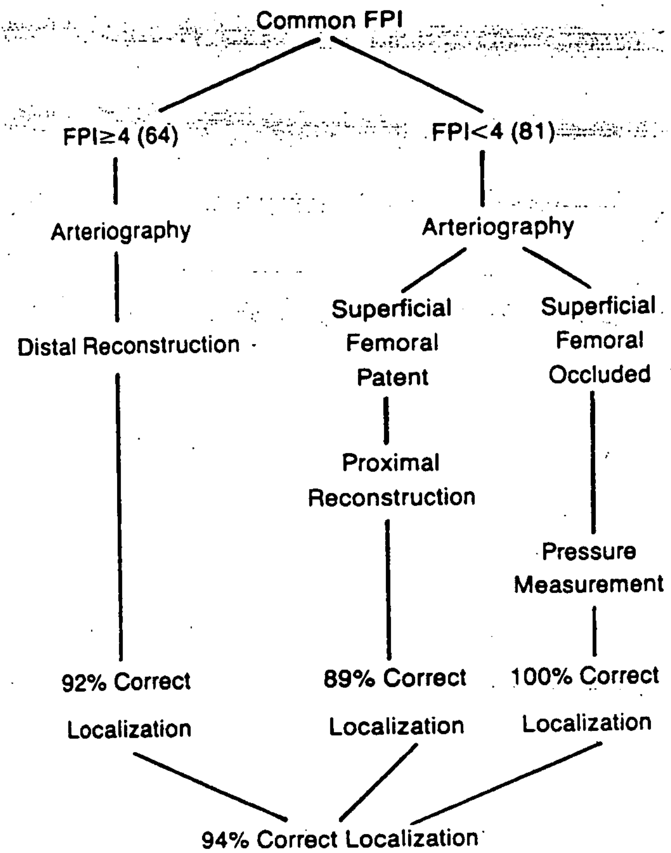


Figure 5–22. Algorithm for localizing hemodynamically significant disease in lower limb arterial segments in claudicants with palpable femoral pulses (n = 145). FPI indicates femoral pulsatility index. (From Thiele BL, Bandyk DF, Zierler RE, et al: Arch Surg *118*:477, 1983. Copyright 1983, American Medical Association.)

correct localization of the disease process in almost all cases (Fig. 5–22).

It seems likely that duplex scanning, which allows direct investigation of the aortoiliac segment, will eventually prove to be the best noninvasive technique for assessing inflow disease (see Chapter 7).[109, 130] The ability to evaluate flow disturbances at the site where they are produced rather than at a considerable distance downstream is a major advantage of this technique. Moreover, infrainguinal disease has little or no effect on local flow disturbances. At present, however, direct femoral arterial pressure measurement remains the gold standard for assessing aortoiliac disease (see Chapter 6).[13, 31, 70, 71, 128] Since pressure measurements are no more invasive than arteriography, are safe, and can be made intraoperatively, they should be obtained whenever there is any question regarding the validity of the noninvasive assessment.[32]

Femorofemoral Bypass

Candidates for femorofemoral bypass present problems analogous to those encountered in patients with multilevel disease. The success or failure of the operation is determined by the functional capacity of the donor iliac artery, which must carry roughly twice the blood flow that it normally does without a significant increase in the pressure gradient from the aorta to the femoral artery.[226] When the donor iliac artery is free of disease, there is no problem, the increased gradient being too small to be recognized. If, however, the donor artery contains atherosclerotic plaques, which it usually does, a minor pressure drop that is tolerable preoperatively may become a major pressure drop postoperatively. Although a modest drop in the resting ankle pressure in the donor limb may be a small price to pay for a significant rise in the ankle pressure of an ischemic recipient limb, a large drop in the postexercise ankle pressure in the donor limb is likely to be unacceptable when the operation has been performed for disabling claudication.[93] Figure 5–23 illustrates this situation in a man with a stenotic donor iliac artery, an occluded recipient artery, and no infrainguinal arterial obstruction.[226] Following construction of a femorofemoral shunt, the ankle pressure response to exercise improved in the recipient limb but deteriorated in the donor limb, becoming essentially identical, and the patient's treadmill walking time decreased from 3 minutes to 2 minutes, indicating a worsening of his claudication.

Harris and associates, reporting on a series of femorofemoral bypasses performed for claudication, found that the ankle pressure responses to exercise deteriorated in 45 per cent of the donor limbs, despite the fact that none of the donor iliac arteries appeared significantly stenotic on arteriography.[93] Of the donor limbs with impaired exercise responses, 90 per cent had normal triphasic femoral arterial velocity waveforms, indicating that this test is not sufficiently sensitive to detect the low-grade lesions responsible for the postexercise pressure drops. In two thirds of the hemodynamically impaired donor limbs, direct femoral artery pressure measurements obtained after injection

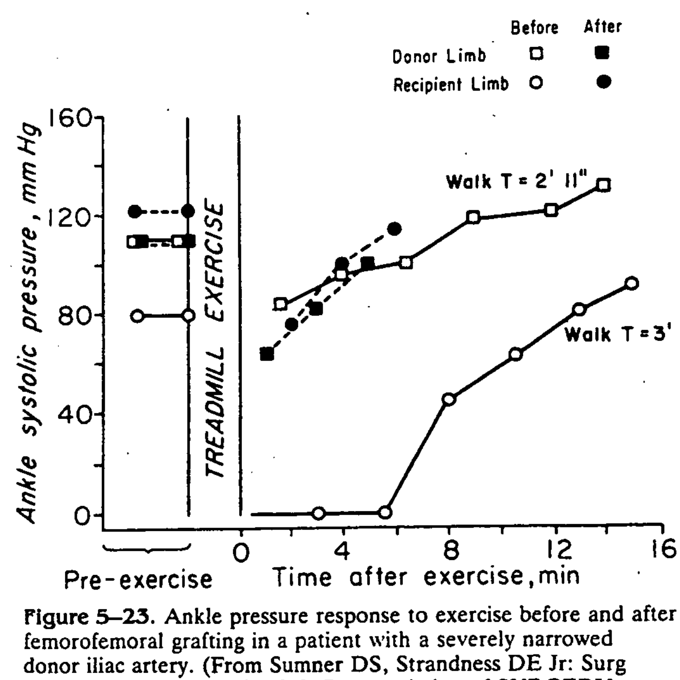


Figure 5–23. Ankle pressure response to exercise before and after femorofemoral grafting in a patient with a severely narrowed donor iliac artery. (From Sumner DS, Strandness DE Jr: Surg Gynecol Obstet *134*:629, 1972. By permission of SURGERY GYNECOLOGY & OBSTETRICS.)

of papaverine also failed to detect an abnormality. It is probable that papaverine, which usually results in a doubling of the blood flow, does not duplicate the stresses imposed by exercise, which may increase blood flow by a factor of five to ten. Nonetheless, direct pressure measurement remains the best physiologic test available for assessing donor limb hemodynamics. In the future, however, it may be supplanted by duplex scanning, which is more sensitive to low-grade lesions.

Other Studies

Regardless of whether noninvasive testing has been used to make the diagnosis of arterial occlusive disease, preoperative studies are of value as a baseline for follow-up evaluation. At a minimum, these should consist of ankle pressure determinations. In patients with severe ischemia (especially diabetics with incompressible ankle vessels), toe pressures and $tcPo_2$ measurements are also valuable. In claudicants, treadmill exercise testing provides an objective method for ascertaining the benefits of the procedure.

Saphenous vein mapping with the duplex scanner is a new and potentially very important adjunctive use of preoperative noninvasive testing (see Chapter 7).[191]

Predicting Success of Operations

Bernstein and associates have stated, ". . . the area in which the noninvasive laboratory has the greatest opportunity to influence patient care lies in its potential as a predictive tool."[24] While some may take issue with this statement, the ability to accurately estimate the likelihood of success or failure of a specific management plan is certainly of great importance.

Sympathectomy

With the advent of reconstructive surgery, there are few indications for lumbar sympathectomy. Treating claudication by means of sympathetic denervation has no physiologic basis (see Chapter 3). Some surgeons, however, feel that it may be beneficial in patients with mild rest pain or superficial ulcers; but this too is open to question. It certainly has no role in the management of the severely ischemic leg. Sympathetic dystrophy and hyperhidrosis remain the only firm indications for the procedure.

The use of digital plethysmography to identify the presence of sympathetic activity and the ability of the peripheral arterioles to dilate in response to sympathetic denervation has been discussed earlier in this chapter.

As one might expect, the success of sympathectomy (in terms of elevating cutaneous blood flow) is related to the local perfusion pressure. With pressures below a certain level, the arterioles are completely dilated and therefore do not respond further to sympathetic denervation. Uhrenholdt demonstrated that pedal blood flow increased in response to a sympathetic block when the skin pressure exceeded 40 mmHg but decreased when the pressure was less than 20 mmHg.[239] Similarly, Thulesius and colleagues, in a series of patients with rest pain or ischemic ulcers, found that the toe temperature increased following a sympathectomy when the ankle pressure was more than 60 mmHg but fell when the pressure was less than 60 mmHg.[234] These results suggest that sympathectomy may result in a steal in limbs with low pedal blood pressures and, therefore, might actually be harmful. In the series reported by Yao and Bergan, 96 per cent of limbs with severe ischemia and ankle pressure indices below 0.21 required amputation following a sympathectomy.[257] When the indices exceeded 0.35, the results were satisfactory. More recently, Walker and Johnston observed that sympathectomy was unlikely to be beneficial in any ischemic limb with an associated neuropathy.[244] In the absence of neuropathy, a successful outcome was likely in limbs with rest pain or digital gangrene, provided the ankle pressure exceeded 30 mmHg. About 50 per cent of limbs with more severe ischemia (forefoot or heel gangrene) were predicted to respond favorably when the ankle pressure was greater than 60 mmHg.

Plecha and associates observed that a distal thigh-arm index greater than 0.7 was associated with 78 per cent good results from sympathectomy and that an index below this level uniformly predicted a poor outcome.[176] Neither the presence or absence of diabetes nor the level of ankle pressure had any discernible effect. In fact, five of six limbs with zero ankle pressures and favorable thigh indices (> 0.7) responded well. Neuropathy was not considered in their study, and no limbs with extensive tissue necrosis, forefoot gangrene, or uncontrolled infection were included. Again, their findings confirm the importance of an adequate inflow to the success of surgical sympathectomy.

In summary, it appears that sympathectomy is likely to be successful only in limbs with ankle pressures greater than 30 to 60 mmHg—in other words, in limbs with low-grade ischemia that would probably respond quite well to judicious medical management. There is little likelihood of success in limbs with low ankle pressures and even some chance that the procedure might be detrimental.

Profundaplasty

When performed for limb salvage, profundaplasty as an isolated procedure is effective in 33 to 86 per cent of cases.[30] To be successful, the operation must be performed on a severely stenotic profunda femoris artery and the profunda-popliteal collateral bed must be well developed. If collateral resistance is too high, the reduction in total limb resistance following profundaplasty will be insufficient to alleviate ischemia. In order to better predict the outcome of profundaplasty, Boren and associates developed an index of popliteal collateral arterial resistance.[30] This index is calculated by dividing the gradient across the knee (above-knee pressure minus below-knee pressure) by the above-knee pressure. When the index was less than 0.25, 67 per cent of the operations were successful; when the

index was greater than 0.50, there were no successful results.

Femoropopliteal and Femorotibial Reconstruction

Since one of the major factors determining the potential for graft survival is the resistance of the recipient vascular bed, it seems logical that the preoperative ankle pressure index should correlate inversely with the incidence of femoropopliteal graft failure.[61]

Dean and colleagues found that 90 per cent of autogenous saphenous vein femoropopliteal grafts inserted in limbs with ankle pressure indices less than 0.20 failed in the early postoperative period.[61] Based on this observation, they suggested that attempting femoropopliteal reconstruction in such limbs may be unwarranted. Others, having had a less dismal experience, are not as pessimistic.[53, 65, 194, 197] The combined early and late failure rates of femoropopliteal bypasses in legs with ankle indices less than 0.2 were 47 per cent in the series reported by Corson and associates[53] and 33 per cent in Samson and coworkers' series.[194] Even when no preoperative ankle pressure can be measured owing to the absence of pedal Doppler signals, many femoropopliteal grafts survive.[65, 197, 247]

While there appears to be a general trend toward better graft survival as the preoperative ankle pressure index increases, the data overlap so extensively that statistical significance is not reached.[53, 61, 65] Thus, the individual ankle pressure index has little prognostic value. In fact, Samson and associates[194] found no difference between the preoperative ankle pressure indices in limbs with grafts that failed and those that remained patent and almost identical failure rates in those with indices below and above 0.2. Although Corson and coworkers observed no statistically significant correlation between failure rate and ankle pressure indices, they did note that an index greater than 0.5 appeared to be a favorable prognostic indicator, since only 3 per cent of the grafts placed under these circumstances failed in the early postoperative period and only 15 per cent failed later.[53] Preoperative PVRs are also of no value in predicting femoropopliteal graft survival.[194]

Since grafts to the distal peroneal, posterior tibial, anterior tibial, and dorsalis pedis arteries bypass the below-knee obstructive disease that may be responsible for the failure of grafts terminating at the popliteal area, ankle pressure indices would not be expected to correlate well with graft survival. In our experience, only 2 of 11 distal bypass grafts in limbs with preoperative ankle indices less than 0.2 failed within 30 days (see Fig. 5–4).[228] Moreover, the average preoperative ankle index of limbs in which grafts failed within 6 months (0.28 ± 0.22) was not significantly different from that of limbs whose grafts remained patent for more than 12 months (0.32 ± 0.26). Although Samson and associates found that the mean preoperative ankle pressure index associated with graft survival (0.37 ± 0.30) was significantly greater than that associated with graft failure (0.27 ± 0.27), there was so much variability that individual values were meaningless.[194] Early failure occurred in 37 per cent of legs with ankle indices less than 0.2 and in 31 per cent of legs with PVRs less than 5 mm; but about 50 per cent of the grafts in legs with no detectable ankle pressure or pulse volume recorded pulses survived. Others have made similar observations.

In summary, preoperative ankle pressure indices and pulse volume recordings are only roughly correlated with infrainguinal graft survival; indices of 0.2 or PVRs of 5 mm are poor discriminators, and many grafts survive in limbs in which no ankle pressures or pulses are detected preoperatively. The decision to perform a bypass graft should not be influenced by noninvasive test results.[53, 65, 194, 197, 228]

Aortofemoral Reconstruction

Aortofemoral reconstructions are functionally successful only when hemodynamically significant disease is present in the aorta or iliac arteries.[192] Accurate assessment of the severity of arteriographically demonstrated lesions may be difficult, especially when there is concomitant involvement of the infrainguinal arteries. Physiologic methods for diagnosing and evaluating the relative importance of disease in these two locations have been discussed earlier in this chapter.

Bernstein and associates followed 80 patients who had undergone aortofemoral bypass grafting to determine the predictive value of a battery of noninvasive tests performed preoperatively.[24] When the ankle index was greater than 0.80, 94 per cent of the limbs became asymptomatic or improved. An index less than 0.40 was associated with a 64 per cent improvement rate. The time required for the toe pulse to regain half its baseline amplitude after a 4-minute period of cuff-induced ischemia (TPRT/2) proved to be the best discriminator. All limbs with a TPRT/2 less than 10 seconds either became asymptomatic or improved, but only 60 per cent of those with a TPRT/2 of 90 seconds were similarly benefitted. Combining these two studies enhanced the predictive value somewhat, particularly in patients with two levels of disease.

The ability to predict minimum increases in the ankle pressure index may be helpful to the surgeon who is debating whether to bypass one segment or two in patients with multilevel disease. Williams and colleagues, using a method originally devised by Noer and associates, predicted postoperative ankle pressure indices in limbs undergoing aortofemoral bypasses by increasing the preoperative ankle index by the same percentage that the directly measured femoral artery pressure/brachial pressure index would be expected to increase if it attained a normal value of 1.0 after bypass.[164, 165, 247] Mathematically, this equates to the ratio obtained by dividing the preoperative ankle pressure index by the preoperative femoral artery pressure index. When the actual postoperative ankle pressure indices were compared with those predicted, a surprisingly high correlation coefficient was obtained ($r = 0.874$). Only 4 per cent of the actual values failed to increase to within 10 per cent of the predicted value, and most were higher than predicted.

These calculations, of course, are not applicable

when the ankle index is zero or extremely low. Although the "additive transmission" method described by Rutherford and colleagues[192] could be used in this situation, it proved to be only 77 per cent accurate for predicting postoperative ankle indices to within ± 0.10.[192] In this method, the estimated increase in the thigh/brachial index is simply added to the preoperative ankle index. Inaccuracies inherent in estimating femoral pressure by the cuff technique were undoubtedly partially responsible for the relatively poor results obtained by these investigators.

Functional Results

Katsamouris and associates reported that a marked increase in the $tcPO_2$ measured preoperatively on the dorsum of the foot after inhalation of oxygen was predictive of symptomatic improvement after reconstructive surgery in ischemic limbs.[122] All limbs showing no change had a poor result. Similarly, Oh and colleagues found that an increase in $tcPO_2$ of 15 mmHg or more occurring on the dorsum of the foot when the patient shifted from a supine to a standing position was a favorable prognostic sign in severely ischemic legs.[168] Following reconstructive surgery 81 per cent of the legs were saved or had patent grafts. When the $tcPO_2$ failed to increase or increased by less than 15 mmHg, only 29 per cent of the legs were salvaged or had patent grafts.

Postoperative Monitoring

Monitoring the patency of an arterial reconstruction once the patient reaches the recovery room is of obvious importance to the successful outcome of the surgical procedure. Assuming that the intraoperative assessment (discussed in Chapter 26) revealed no abnormalities, the surgeon is obligated to detect early signs of graft failure in order to correct the responsible defect as expeditiously as possible. The problem is likely to be technical but may be a thrombosis for which there is no apparent explanation. Early correction of a minor problem often ensures long-term patency.

The reappearance of peripheral pulses in limbs in which the pulse was not palpable preoperatively is good evidence of graft patency, and the disappearance of pulses palpable in the operating room at the termination of the procedure is highly suggestive of occlusion. But pedal pulses may be weak immediately after operation and may be absent in limbs with residual distal disease. Although the presence of a palpable pulse in a graft is reassuring, it does not necessarily imply patency. Despite the absence of blood flow, a graft can continue to pulsate when its terminal end is occluded as long as the column of blood remains liquid or is incompletely thrombosed. Furthermore, it is seldom possible to palpate graft pulsations when the graft is placed deeply within the leg.

The Doppler flowmeter is the most versatile noninvasive instrument for assessing graft patency in the immediate postoperative period (Fig. 5–24). The presence of pulsatile, often hyperemic flow clearly establishes patency. Absence of a velocity signal or the presence of an abrupt signal with a thumping quality indicates occlusion even when the graft may continue to pulsate. Unfortunately, it is frequently impossible to detect a flow signal through a polytetrafluoroethylene graft soon after it has been placed, owing to the severe damping of ultrasound by air trapped in the interstices of the graft material. Later, as these areas are infiltrated by body fluids, the signal is readily detected. There is no problem in investigating grafts of autogenous vein, umbilical vein, or Dacron. It is, however, difficult to examine aortofemoral grafts at groin level because of the fresh incision, depth of the graft, and the disruption of tissue planes, all of which interfere with the transmission of ultrasound.

Signals from the dorsalis pedis, posterior tibial, and anterior medial tarsal arteries should be assessed in all patients. It must be emphasized that the mere presence of an audible signal does not connote graft patency. In the hands of an experienced observer, the quality of the Doppler signal can, nevertheless, be quite informative. For example, hyperemic flow or a normal triphasic signal is excellent evidence of graft patency. On the other hand, a barely audible, nonpulsatile signal or the absence of a signal in a pedal artery that was patent preoperatively and is known to be in continuity with the reconstruction must be viewed with concern. Immediately after proximal (aortoiliac, femorofemoral, or axillary femoral) reconstruction in limbs with associated femoropopliteal occlusion, flow signals may not be detected at the ankle.[256] In this event, an audible popliteal signal signifies patency of the proximal reconstruction. If the pedal flow signals remain inaudible 4 to 6 hours after the operation, the possibility of thrombosis must be entertained.

Measurement of ankle pressure can be accomplished in all cases with an audible pulse except those in which the terminal anastomosis is at foot level or those in which peripheral incisions make the examination too painful. An increase in the ankle-brachial

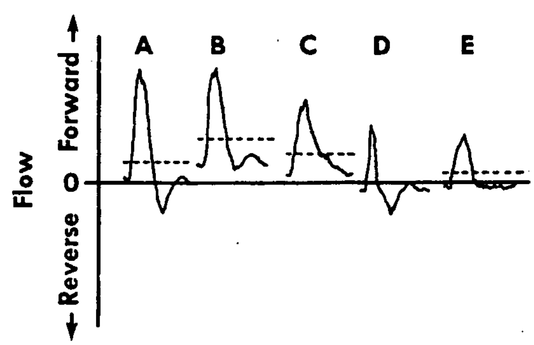


Figure 5–24. Contour of flow pulse in a vein graft. Dotted line indicates mean flow. *A*, Unobstructed graft with peripheral vasoconstriction. *B*, Unobstructed graft with peripheral vasodilation. *C*, Stenosis proximal to the site of the flow probe. *D*, Complete obstruction several centimeters distal to site of flow probe. Note that the pulse contour is nearly normal but mean flow is zero. *E*, Stenosis just below site of flow probe.

index of more than 0.15 compared with the preoperative value is indicative of graft patency.[195] Ordinarily, the increase will be appreciably greater than this. Failure to demonstrate an increase in the ankle index suggests, but does not conclusively establish, the existence of a problem. If other evidence is compatible with continued graft function, it is usually safe to delay arteriography or re-exploration for a variable period of time (depending on the type of operation) while the ankle pressure is carefully monitored.[256] The limit should be no more than 4 hours following a femoropopliteal bypass. If, after that time, the ankle pressure does not improve, the patient should be returned to the operating room for correction of the problem. In limbs with chronic obstructions below the site of the distal anastomosis, it may take much longer for the ankle index to rise. For example, improvement may not be apparent for 4 to 6 hours following a bypass to a blind popliteal segment, for 6 to 12 hours after an aortofemoral bypass in a limb with multilevel disease, and for 24 hours after a profundaplasty.[256]

Pulse volume recordings may be useful in limbs in which ankle pressures cannot be obtained either because of arterial calcification or because of painful incisions.[60, 195] An increase of more than 5 mm in the amplitude of the ankle PVR tracing compared with that obtained preoperatively is indicative of graft patency. A decrease in the amplitude is strongly suggestive of obstruction.

Follow-up Studies

After the immediate postoperative period, noninvasive studies continue to be important, affording an objective assessment of improvement and permitting failure or impending failure of the reconstruction to be detected.

Assessing Results of Surgery

Ankle pressure measurements provide the most convenient method of evaluating the results of reconstructive arterial surgery. An increase of 0.15 in the resting ankle pressure index can be taken as definite evidence of improvement. If all involved segments have been bypassed, the result should be an ankle index of 1.0 or more, provided the diameter of the graft is sufficiently large to carry the required flow with a minimal pressure gradient. During the hyperemic phase following revascularization of a severely ischemic extremity, ankle indices, though improved, may remain low when the graft diameter is less than 3.5 mm.[12] This situation is most likely to be encountered when in-situ grafts terminate far distally in the leg, but it may also occur in association with femoropopliteal bypasses. As the hyperemia decreases, the ankle pressure gradually rises, perhaps requiring a month before reaching a maximum level. If, however, all diseased arterial segments have not been bypassed or otherwise reconstructed, the ankle pressure index will rise commensurate with the reduction in total resistance but will not attain normal levels.[164, 192, 247] Many examples of

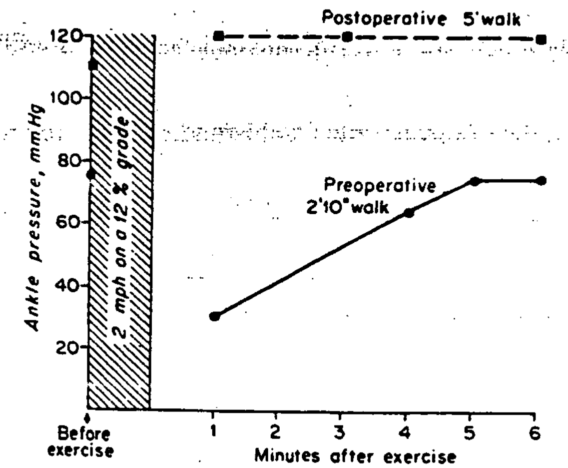


Figure 5–25. Resting ankle pressure and ankle pressure response to exercise in a 50-year-old man with calf and thigh claudication due to occlusion of left common iliac artery. After bypass grafting, ankle pressure and ankle pressure response to exercise returned to normal. The patient was relieved of symptoms. (From Strandness DE Jr: J Cardiovasc Surg *11*:192, 1970.)

this, including aortofemoral bypasses in limbs with superficial femoral obstruction and bypasses to a blind popliteal segment, occur.

Unfortunately, as repeatedly emphasized in this chapter, ankle pressure measurements may be unreliable in 5 to 10 per cent of revascularized limbs, owing to medial calcification of the peripheral arteries. In addition, the presence of pedal arterial disease may continue to restrict perfusion of the foot despite adequate inflow pressures. Pulse volume recordings at ankle or foot level, digital plethysmography, measurement of toe pressures, and $tcPo_2$ determinations are often quite helpful for assessing functional results in such cases.[122, 132, 135]

When surgery has been performed to alleviate claudication, exercise testing affords a sensitive means of judging physiologic improvement. In the ideal case, the patient will be able to walk for 5 minutes without experiencing a drop in ankle pressure (Fig. 5–25).[212] When residual disease is present or when the diameter of the graft restricts flow, there will be a postexercise pressure drop even though the resting ankle index is normal and the patient walks for 5 minutes without developing claudication (Fig. 5–26).[216] Other patients may be symptomatically improved, walking for a longer time on the treadmill with a less severe drop in ankle pressure, but will be far from normal. This result is commonly seen when arterial reconstruction is limited to the aortofemoral segment in patients with multilevel disease (Fig. 5–27).[213]

Obtaining a baseline study early after reconstruction is important for further follow-up. Patient testimony and the physical examination may be unreliable.[212] Often, the ankle pressure and exercise tolerance continue to increase for several days to several months, possibly in response to the increased head of pressure made available by a proximal reconstruction.[258] Although the explanation is not readily apparent, many patients with femoropopliteal bypasses also improve.[217, 258]

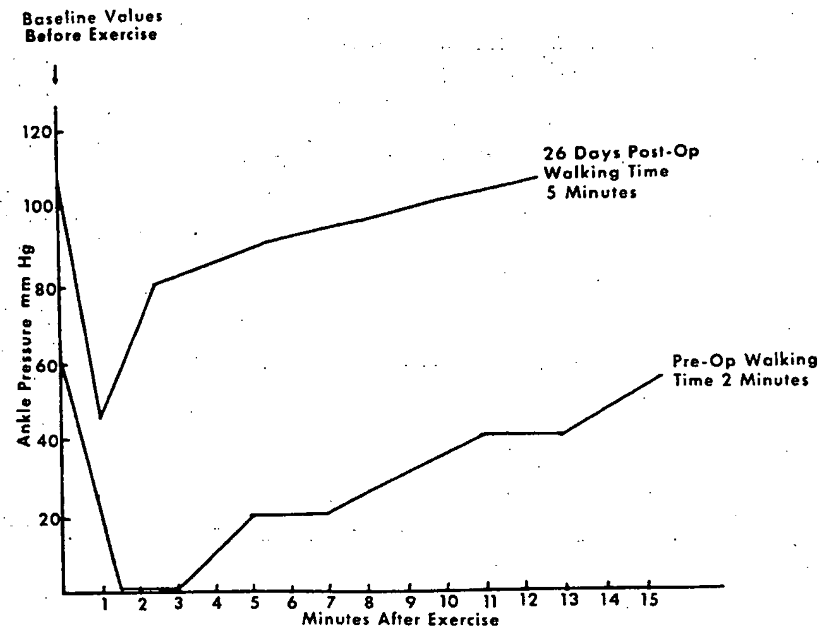


Figure 5–26. Effect of exercise on ankle pressure response before and after a "successful" femoropopliteal bypass graft. Following the operation, the patient regained his popliteal and pedal pulses and became asymptomatic. His resting ankle pressure returned to normal levels. Although he could walk five minutes on the treadmill without experiencing claudication, his ankle pressure response to exercise remained abnormal. (From Strandness DE Jr: Surgery *59*:325, 1966.)

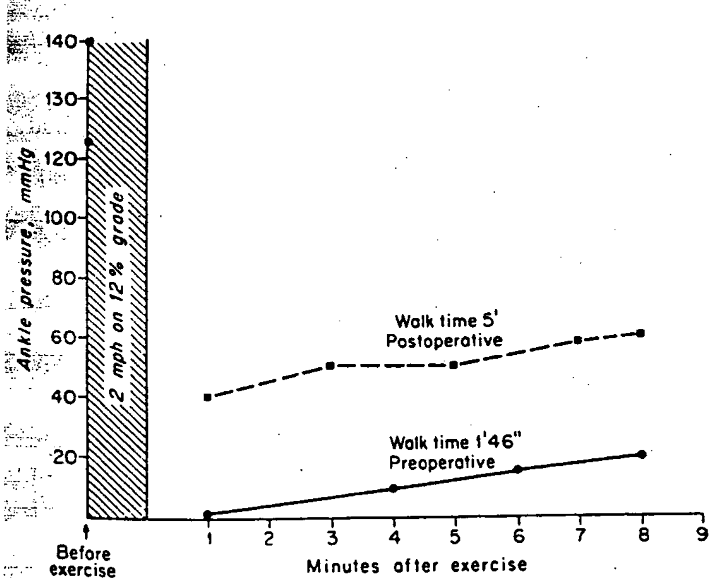


Figure 5–27. Effect of aortofemoral grafting on exercise response in a patient with combined iliac stenosis and superficial femoral occlusion. Walking time increased greatly, but ankle pressure response to exercise, though improved, remained abnormal. (From Strandness DE Jr: *In* Dale WA (ed): Management of Arterial Occlusive Disease. Chicago, Year Book Medical Publishers, 1971, pp 13–29.)

Predicting Early Failure of Bypass Grafts

Corson and colleagues observed that 88 per cent of vein bypass grafts to the popliteal, tibial, or peroneal arteries failed in the early (< 30 days) postoperative period when the postoperative ankle index was less than 0.50 and that 91 per cent of early failures had postoperative indices below 0.70.[53] When the increase in the postoperative index compared with that obtained preoperatively was less than 0.10, 88 per cent of the grafts failed early. All early failures were in limbs in which the increase in the index was less than 0.40. Samson and associates, on the other hand, found that neither the absolute value of the postoperative ankle index, the PVR amplitude, nor the increase in these two measurements had any predictive value.[194] Other investigators have reached similar conclusions.[65, 228]

Bandyk and colleagues, who used pulsed-Doppler ultrasound to examine blood flow in in-situ vein bypass grafts, found that a peak systolic velocity less than 40 cm per second detected technical errors.[12] Even in the absence of an identifiable error, low velocities predicted early failure. Owing to the hyperemia that follows successful arterial reconstruction, diastolic forward flow is typically present and is a good prognostic sign. Absence of diastolic forward flow indicates a high peripheral resistance, an observation that correlates with early failure.[11, 12]

Recognition of Impending Graft Failure

Stenoses or other problems that jeopardize graft survival are best corrected prior to the development of thrombosis.[21, 22, 49, 241] Once a graft has thrombosed, successful thrombectomy does not ensure long term patency, even when appropriate measures have been taken to rectify the responsible defect.

Stenoses may develop in femoropopliteal or femorotibial grafts without producing any symptoms or any alteration of the pulses in the grafts or at the periphery.[21, 22] Many of these "silent" stenoses can be detected if the ankle pressure is followed closely in the months and years after operation.[22, 49, 170, 228, 237, 241] Close surveillance is particularly important during the first year. since about three quarters of the problems arise within this early period. A previously stable ankle pressure index that drops by 0.15 or more (or a decrease in the PVR amplitude of 5 mm or more) suggests the need for arteriographic investigation.[7, 21, 22, 11[illegible], 237] As a rule, operative correction is easily accomplished, after which the ankle pressure should return to the original postoperative level.

Unfortunately, in an appreciable number of limbs, no drop in ankle pressure will be evident prior to graft failure.[11, 252] For this reason, Bandyk and coworkers recommend following the velocity of blood flow in recently implanted grafts as a supplement to ankle pressure measurements.[11] A decrease in systolic flow velocity below 40 cm per second has, in their experience, proved to be an ominous sign. Diastolic flow velocity, on the other hand, has little predictive value. It normally decreases in the weeks following graft insertion—essentially disappearing by 3 months—at which time, a typical triphasic flow pattern is established (see Fig. 5–24*A*).[12]

The function of a graft and its longevity may also be compromised by the progression of atherosclerotic or fibrodysplastic disease at the anastomoses or in the inflow or outflow arteries.[228, 237] Again, these changes, which may not be clinically apparent, can often be detected by a fall in the ankle pressure index (Fig. 5–28). Segmental pressures may help to distinguish stenoses developing in a bypass graft or endarterectomized segment from disease progression in arteries above or below the reconstruction.[154, 155]

Duplex scanning is potentially a powerful method for following bypass grafts.[11, 57, 139, 150] Not only can the presence of a graft-threatening defect be detected but also the problem can be localized to the inflow or outflow arteries, the anastomoses, or to the graft itself. Moreover, stenoses can be identified before they become hemodynamically significant, that is, before the ankle index begins to fall or before exercise test results become abnormal.[11, 150] Although the natural history of these early lesions has not been established, it seems logical to predict that many, if not most, will progress.[150, 252] Duplex scanning also facilitates the recognition of residual arteriovenous fistulas and valve leaflets, problems unique to in-situ bypass grafting.[57, 139] In addition, aneurysms involving the graft, anastomotic false aneurysms, and perigraft seromas are easily evaluated (see Chapter 7).

Hemodynamic Failure

Continued patency of a bypass graft or endarterectomy has been the traditional standard for success of reconstructive surgery. The reconstruction may remain patent, however, without providing functional improvement or relief of symptoms.[170, 192, 227] If patent but hemodynamically failed grafts are recognized, the condition responsible for the lack of physiologic improvement may be susceptible to correction. Any lesion within the graft or inflow or outflow arteries can be responsible.[154, 155, 170] Of particular interest is the occurrence of hemodynamic failure in the absence of any new or residual lesion. For example, the diameter of the graft may merely be too small to permit a flow rate adequate to relieve symptoms.[170] Segmental pressure studies, Doppler surveys, exercise testing, and duplex scanning are all valuable methods for identifying and evaluating the severity of hemodynamic failure (Fig. 5–29).

Directional Doppler flow studies are especially helpful in the evaluation of the functional results of femorofemoral bypass grafting (see Fig. 5–23).[226] Of 51 such grafts examined by O'Mara and coworkers, two were found to have flow going in the opposite direction to that originally intended.[170]

Natural History and Prognosis

Symptoms and physical signs are too unreliable and arteriography is too invasive to be used to study the

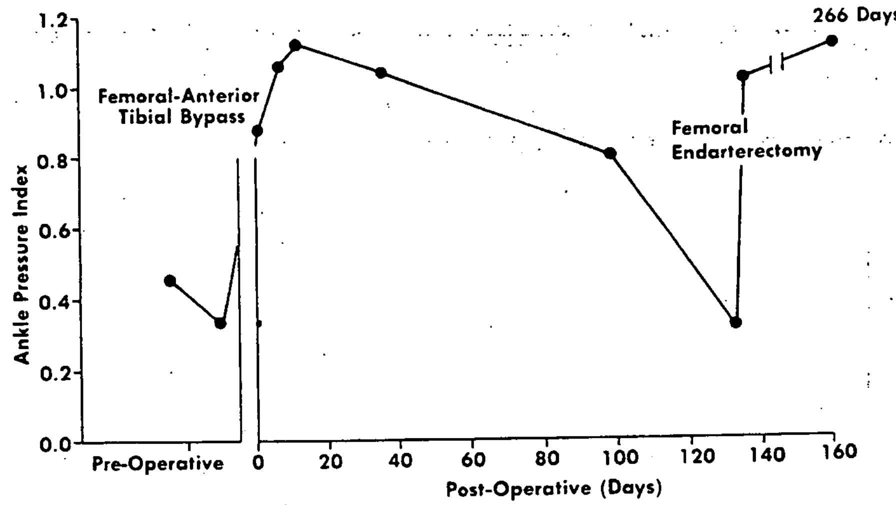


Figure 5–28. Salvage of femoral anterior tibial graft prior to failure. The drop in ankle pressure following a femoral anterior tibial bypass called attention to the development of a stenosis in the common femoral artery above the proximal anastomosis. An endarterectomy restored normal function. (From Sumner DS, Strandness DE Jr: Surgery *86*:442, 1979.)

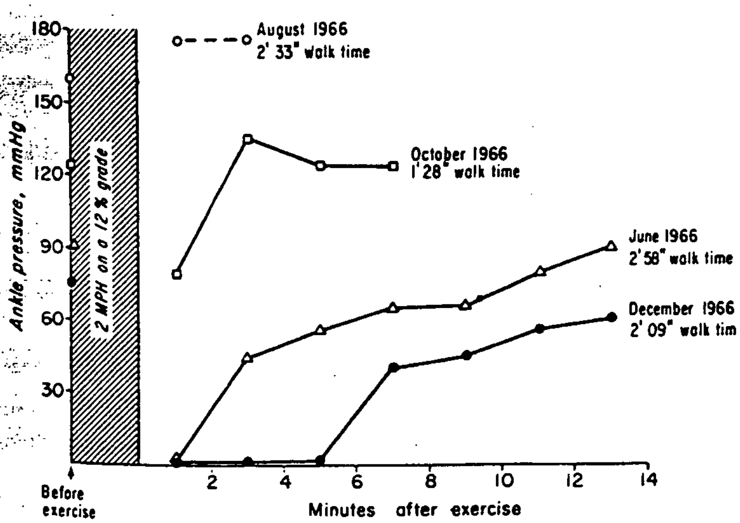


Figure 5–29. Changes in ankle pressure and postexercise response in a 65-year-old man who underwent an aortic bifurcation graft in June 1966. In August 1966, ankle pressure was normal and there was no drop in pressure following exercise. By October 1966, however, the exercise response was abnormal, and by December 1966, the objective tests were worse than prior to operation. Although the graft remained patent, the patient developed an occlusion in the superficial femoral artery. (From Strandness DE, Jr: Techniques in the evaluation of vascular disease. *In* Cooper P, Nyhus LM (eds): Surgery Annual, Norwalk, CT, Appleton-Century-Crofts, 1971, pp 181–197.)

prevalence, incidence, and natural history of peripheral arteriosclerotic occlusive disease.[55,144] Because of their objectivity and ability to be repeated frequently and indefinitely without subjecting the patient to undue discomfort or risks, noninvasive tests are ideally suited for population surveys and longitudinal studies of disease progression.

Criqui and coworkers reported that only 1.9 per cent of a geographically defined population ranging in age from 38 to 82 years (mean, 66 years) complained of intermittent claudication, but noninvasive testing of this same population revealed a 11.7 per cent prevalence of large vessel peripheral arterial disease.[54] The prevalence ranged from less than 3 per cent in subjects under the age of 60 years to more than 20 per cent in subjects older than 75 years. Identification of any symptom that could possibly be called claudication or the recognition of any pulse abnormality had a positive predictive value of only 37 per cent. Similarly, Marinelli and colleagues found that 165 (36 per cent) of 458 diabetic patients had positive noninvasive test results, indicating the presence of hemodynamically significant peripheral arterial disease.[144] Nearly one third of the patients with no history of intermittent claudication and one fifth of those with normal physical examinations had abnormal test results. Using noninvasive methods, Strandness and Stahler detected disease progression in 52 per cent of patients with lower extremity arterial disease who were followed for an average of 3 years.[219] Almost 40 per cent of patients with disease progression experienced no increase in symptoms. Clearly, clinical evaluation alone underestimates both the prevalence of arteriosclerosis obliterans and its rate of progression.

Based on a 5-year follow-up study of nondiabetic patients with intermittent claudication due to occlusion of the superficial femoral artery, Wilson and associates concluded that symptoms are likely to improve or remain unchanged when the ankle pressure index exceeds 0.60 but are likely to progress when the index is less.[248] Of those patients whose symptoms improved, only 46 per cent demonstrated any objective increase in the ankle index. With more severe disease, the outcome may become evident over a much shorter period of time. Mazza and coworkers found that 57 per cent of limbs with an ankle pressure index between 0.30 and 0.50 could be managed nonoperatively, whereas 92 per cent of limbs with ankle indices less than 0.30 required reconstruction or amputation within 2 years.[148] Paaske and Tønnesen found that 82 per cent of patients with a toe/brachial pressure index less than 0.07 underwent a major amputation within 2 years, and 27 per cent died.[173] The amputation and death rates were 38 and 33 per cent, respectively, in those with toe indices of 0.08 to 0.13 and were 27 and 18 per cent, respectively, in patients with toe indices of 0.14 to 0.25. In our experience, an ankle index less than 0.30 is associated with a 32 per cent amputation rate and a 60 per cent death rate over a 6-year period.[51] This contrasts with a 13 per cent amputation rate and 33 per cent death rate in patients of similar age (72 ± 11 years) with ankle indices between 0.30 and 0.50 ($p < 0.05$).

Not all investigators have been able to confirm a strong association between the initial ankle index and symptom progression in limbs with intermittent claudication. For example, Cronenwett and colleagues found that the values overlapped too extensively to establish valid criteria for predicting outcome.[56] Most studies, however, do suggest that noninvasive tests have prognostic value and confirm the association between disease progression, limb loss, early death, and the severity of the atherosclerotic process.

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Uncover the underlying causes

Peripheral arterial disease results in the manifestation of many serious conditions, including critical limb ischemia (CLI). CLI is the end-stage of lower extremity PAD in which severe obstruction of blood flow results in ischemic rest pain, ulcers and a significant risk for limb loss.

National Healing Corporation's evidence-based Clinical Pathway ensures that the appropriate processes and procedures are put into place to identify nonhealing wounds caused by CLI. Appropriate diagnosis and care is crucial, especially given the variety of complications that can occur with chronic wounds.

This edition of *Wound Healing Perspectives* is dedicated to covering the various etiologies and underlying causes associated with CLI, including diabetes, PAD, cigarette smoking, hypertension, hypercholesterolemia, family history, and lifestyle. We also review non-invasive treatments for CLI, diabetic foot ulcers, hyperbaric oxygen therapy for CLI patients, and prevention strategies for amputation.

As always, we appreciate the opportunity to bring you interesting and informative insights. We hope you enjoy this installment of *Wound Healing Perspectives.*

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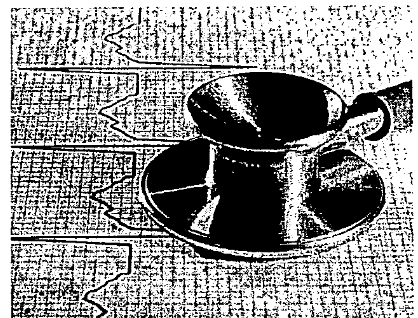
Critical limb ischemia:
Overview and treatment options

Critical limb ischemia (CLI) is the term used for patients with chronic ischemic rest pain, ulcers, or gangrene attributed to inadequate blood flow or arterial occlusive disease. It is the progressive evolution and clinical manifestation of peripheral arterial disease (PAD). CLI differs from acute limb ischemia, which generally follows arterial thrombosis or peripheral thromboembolism.

Patients with CLI often suffer from severe pain caused by ischemia, tissue loss, ischemic neuropathy, or a combination of these factors. The pain typically occurs at night when the patient is resting, and the episodes can last hours. Controlling the pain of CLI patients, therefore, is very important and is often achieved through reperfusion of the affected limb or administering large doses of analgesics (e.g. acetaminophen) on a regular basis, nonsteroidal anti-inflammatory drugs, narcotics, or opiates (Diehm and Diehm, 2004). Physicians should assess the severity of a patient's pain through use of pain scales or visual scales. (See Leriche-



CLI PATIENTS ARE AT A HIGH RISK OF MYOCARDIAL INFARCTION, STROKE, AND VASCULAR DEATH.

Fontaine classifications on page 2).

A large percentage of patients with CLI have coexisting diseases, such as cardiovascular and renal disorders. Furthermore, since many CLI patients are

(continued on page 2)

HIGHLIGHTS INSIDE

Critical limb ischemia *(continued from page 1)*

smokers, lung diseases such as chronic bronchitis and bronchial carcinoma also are prevalent in this population (Diehm et al, 2004). CLI patients also are at high risk for myocardial infarction, stroke, and vascular death. Therefore, prompt referral to a specialized vascular center improves the success of their treatment and reduces the systemic risk in this population (Diehm et al, 2004).

Leg revascularization procedures work well for patients with CLI (or those with disabling claudication) by providing sufficient blood flow to relieve rest pain and heal skin lesions. If not revascularized, patients with CLI can lose limbs or acquire other potentially fatal complications due to gangrene progression or sepsis (Novo, Coppola and Milio, 2004).
For patients suffering from high-grade stenoses or short arterial occlusions, percutaneous transluminal angioplasty (PTA) is typically recommended as the first form of treatment. In those patients where amputation is not required and vascular reperfusion is not possible when using thrombolysis, angioplasty, or surgical reconstruction, medical treatment to improve microcirculatory blood flow should be considered (Diehm et al, 2004).

Surgical procedures—preferably endovascular techniques—are second on the list, offering patients a lower morbidity and mortality risk compared to open surgical revascularizations (Novo et al, 2004). In fact, advances in vascular and endovascular procedures in recent years provide a much better chance of limb salvage for many patients. As a result, patients who are at risk for CLI should be diagnosed early and treated promptly.

Approximately 20-30% of CLI patients are not considered candidates for vascular or endovascular procedures, and therefore, amputation is often the only option. Primary amputation also is considered when there is an absence of distal vessels, especially in the case of advanced distal ischemia associated with a low Ankle-Brachial Index (ABI) value (<0.3) (see chart to the right) (Diehm et al, 2004). Because of the occurrence of CLI in patients with PAD, approximately 160,000 amputations are

(continued on page 3)

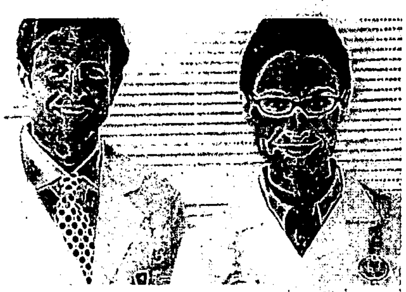
The following chart defines the ischemic rest pain of patients without skin lesions and the presence of ulcers or gangrene.

LERICHE-FONTAINE CLASSIFICATION

STAGES	SYMPTOMS	PATHOPHYSIOLOGY	PATHOPHYSIOLOGY CLASSIFICATIONS
Stage I	Asymptomatic or effort pain	Relative hypoxia	Silent arteriopathy
Stage IIA	Effort pain/pain-free walking distance > 200 m	Relative hypoxia	Stabilized arteriopathy, non-invalidant claudication
Stage II B	Pain-free walking distance < 200 m	Relative hypoxia	Instable arteriopathy, invalidant claudication
Stage III A	Rest pain, ankle arterial pressure > 50 mm Hg	Cutaneous hypoxia, tissue acidosis, ischemic neuritis	Instable arteriopathy, invalidant claudication
Stage III B	Rest pain, ankle arterial pressure < 50 mm Hg	Cutaneous hypoxia, tissue acidosis, ischemic neuritis	Instable arteriopathy, invalidant claudication
Stage IV	Trophic lesions, necrosis or gangrene	Cutaneous hypoxia, tissue acidosis, necrosis	Evolutive arteriopathy

SOURCE: NOVO ET AL, 2004

CLI non-invasive treatments



Many non-invasive treatments are available for patients with CLI and are revolutionizing the way they are treated. These include:

- **Balloon angioplasty and stenting:** for focal segments of narrowing or short occlusions of the iliac arteries. However, according to Novo et al (2004), recent reports reveal that percutaneous transluminal angioplasty or more complex endovascular procedures (e.g. excimer laser recanalization followed by balloon angioplasty) may be useful in treating arteries below the knee.
- **Cryoplasty:** a form of angioplasty that simultaneously dilates and cools the plaque and vessel wall in the treatment area and essentially opens leg

(continued on page 3)

performed annually in the United States (Biamino, 2004).

Contradictory studies on aggressive revascularization, however, do exist. Tunis et al (1991) reported that in the United States the increased use of interventional procedures (e.g. angioplasty, including stenting implant) between 1979-1989 did not result in fewer amputations. The rate of amputations, in fact, increased from 1 to 24 per 100,000 cases as did bypass surgery from 32 to 65 per 100,000 cases. Conversely, Novo et al (2004) reported that a Swedish study revealed that over an eight-year period there was a decrease from 42% to 27% in primary amputations associated with a corresponding increase in revascularizations, resulting in an overall decrease in amputation from 61% to 47%.

ANKLE-BRACHIAL INDEX VALUES AND CLINICAL CLASSIFICATION

Normal	> 0.90
Claudication	0.50-0.90
Rest Pain	0.21-0.49
Tissue Loss	Trophic lesions necrosis or gangrene

Diehm et al (2004) also reported that amputation rates are highly correlated with persistent cigarette smoking. In two series, the amputation rate was between 11% and 23% in those who

TO REDUCE THE RISK OF ADVERSE CARDIOVASCULAR EVENTS, OR EVEN DEATH, PATIENTS WITH CLI ARE URGED TO STOP SMOKING AS WELL AS SEEK PROPER TREATMENT FOR HYPERTENSION, DIABETES, AND HYPERLIPIDEMIA.

continued to smoke, versus 0 to 10% in those who refrained from smoking. In fact, even patients with severe end-stage PAD can benefit from smoking cessation (Diehm et al, 2004).

Risk factors for the development of CLI include age, smoking, and diabetes mellitus. Diabetic patients are 10 times more likely to need a major amputation than non-diabetic PAD patients, so patients are urged to aggressively control their diabetes and maintain fasting blood sugars below 120 mg/dL and post prandial sugars $\leq$ 180 mg/dL, according to Diehm et al. Chronic management should aim at normalizing glycohemoglobin levels to less than 7% (Diehm et al, 2004). What's more, major amputation is more frequent among PAD patients who smoke heavily. To reduce the risk of adverse cardiovascular events, or even death, patients with CLI are urged to stop smoking as well as seek proper treatment for hypertension, diabetes, and hyperlipidemia. Smoking cessation also improves the viability of graft patencies for both vein and prosthetic graft material (Diehm et al, 2004).

Non-invasive tests to properly diagnose patients with PAD (as well as ascertain the severity of the limb ischemia) include measurement of the ankle/brachial index or the ankle and toe Doppler pressures. The detection of transcutaneous PO_2 and PCO_2 and diagnostic imaging studies are also effective tests. It is important to note that diabetic patients often have falsely elevated ABI readings of over 1.3 (Seiman, 2000). ■

CLI non-invasive treatments *(Continued)*

arteries clogged with plaque so that patients can avoid or postpone amputation (Biamino, 2004).

- **Pharmacotherapy (e.g. prostanoids):** helps blood flow in CLI patients and could save 40% of lives and limbs. However, drugs will not replace surgery in CLI patients since surgery saves approximately 60% of limbs (Diehm et al, 2004).
- **Topical therapies and hyperbaric oxygen treatment:** appropriate when revascularization has failed or is not technically possible. Reports on the use of hyperbaric oxygen in patients with early gangrene revealed that pain relief was obtained and amputations could be postponed. ■

General advice for CLI patients

- Patients should inspect feet daily, using mirror if necessary (especially between toes, pressure areas)
- Patients should avoid trauma to the endangered part of the limb
- Patients should take medications regularly
- Patients should avoid pressure in any part of the limb and swelling of the leg (edema)
- Patients should have their blood pressure, blood sugar, and blood lipids checked regularly
- Patient should wear appropriate footwear once feet are healed ■

SOURCE: DIEHM ET AL, 2004.

Peripheral arterial disease:
The forgotten risk factor

Peripheral arterial disease (PAD) is atherosclerosis and artherothrombosis of the leg arteries. The primary symptom, known as intermittent claudication (IC), is pain in the calves on exertion caused by inadequate blood flow to the muscles due to narrowing or blockage of the arteries. Although seemingly innocuous, PAD is a serious clinical problem, potentially life-threatening and often goes undiagnosed since most patients are asymptomatic.

Understanding the patient's medical history combined with the use of non-invasive tests that measure the ankle-brachial index (ABI) help in the diagnosis of PAD.

PAD IS A SERIOUS CLINICAL PROBLEM, POTENTIALLY LIFE-THREATENING, AND OFTEN GOES UNDIAGNOSED (BULL 2005).

The APBI can be calculated from the pressure in the ankle vessels and the brachial pressure—a reading of <0.9 mm Hg indicated the presence of PAD and <0.4 mm Hg indicates severe disease.

- Patients with symptomatic PAD
  - have a 30% risk of death within five years, increasing to almost 50% within 10 years and
  - are 60% more likely to die from a heart attack and 12% more likely from ischemic stroke.
- More than 61% (16.5 million) of PAD sufferers are asymptomatic.
- Hypertension and hyperlipidemia were less likely to be treated in patients with PAD
- Antiplatelet therapy was described for a little more than half of patients with PAD (54%), compared with 71% of CVD patients.
- Smoking is the main risk factor for PAD.
- Other risk factors include diabetes, hypertension, hypercholesterolemia, family history, and lifestyle factors such as obesity, smoking, and leading a sedentary lifestyle.
- Risk factors should be addressed by tight control of HbA1c levels in patients with diabetes, reducing hypertension, and hyperlipidemia.
- Walking is recommended for patients with PAD.
- Men are at higher risk for getting PAD.
- People with PAD are six times more likely to die from cardiovascular disease. ■

SOURCE: BULL, 2005.

Diagnosing peripheral arterial disease:
Costs and impact on medical care

Peripheral arterial disease (PAD), a common disease in the elderly (5-10% are believed to experience PAD), is increasingly being recognized as an indicator of disseminated athero-thrombosis which can lead to myocardial infarction. As a result, there has been a greater interest among the medical community in not only alleviating the symptoms of the disease, but treating the condition itself. Treatment includes using antiplatelet agents as well as other drugs. (Migliaccio-Walle, Caro, Ishak and O'Brien 2005).

Although there is a lot of information on the burden and impact of myocardial infarction, little information is available on the impact of PAD on resource utilization and costs (Migliaccio-Walle et al, 2005). A recent study by Migliaccio-Walle et al examined the impact of a PAD diagnosis on resource utilization and costs by studying a group of Canadian patients with the disease for more than a decade. The study also provides the basis for examining and understanding the economic implications of emerging treatments of the disease. By treating risk factors and implementing preventive measures, the occurrence of cardiovascular events will be reduced thus diminishing costs.

In the study, according to Migliaccio-Walle et al, (2005) hospitalizations and physician visits were categorized into two groups—all-cause and cardiovascular disease (CVD)-related. Procedures were divided into angiography, coronary artery bypass graft, percutaneous transluminal coronary angioplasty, and PAD-related procedures such as amputation, embolectomy, or arterial surgery, for example. Hospitalization as a result of bleeds were a part of this cost analysis.

Corresponding inpatient care unit costs for CVD- and bleed-related diagnoses were used and these costs were then applied on a patient-by-patient basis to the resources consumed in each period (Migliaccio-Walle et al).

PAD ACCOUNTED FOR 1.43 HOSPITALIZATIONS PER PATIENT PER YEAR ON AVERAGE FROM 1985 - 1995. (MIGLIACCIO-WALLE ET AL, 2005).

Hospitalizations
In terms of hospitalizations, for those diagnosed with PAD, 10.7% were hospitalized about 1.08 times in the first month following diagnosis or 1.43 hospitalizations per patient per year. Furthermore, among those PAD patients, less than 20% of all hospitalizations were related to CVD with the greatest proportion (19.3%) occurring in the second month after diagnosis (Migliaccio-Walle et al, 2005).

Length of stay
The average length of hospital stay for PAD patients was 11.6 days but length of hospital stays ranged from as low as 8.7 days to as high as 45 days. Patients who were hospitalized for a bleeding event were hospitalized on average, the longest, followed by CVD-related events.

Costs
The total cost for related hospitalizations (CVD, PAD, and bleed) following a PAD diagnosis ranged from $4.5 million (Canadian dollars) in year nine to $13.3 million (Canadian dollars) in year one and the average cost per patient hospitalized as a result of CVD is approximately $76,151 (Canadian dollars). ■

Health-economic consequences of diabetic foot ulcers

Diabetic foot ulcers and amputations result in huge societal costs and high costs for individual patients. Topical wound treatments and inpatient care account for the largest fraction of costs over time until the patient is completely healed. Costs of materials, staff, and transportation, as well as frequency of dressing changes, the rate of healing, and the final outcome are factors that can effect the total costs and cost effectiveness of topical treatments.

The major costs for infected diabetic foot ulcers that healed after an amputation occur between amputation and complete healing and are mainly related to topical treatments. Total direct costs for healing infected ulcers not requiring amputation are approximately $17,5000 (US dollars) compared to lower-extremity amputation which typically range from $30,000-33,500. Prevention of foot ulcers and amputation, then, is the best cost-saving strategy.

SOURCE: RAGNARSON TENNVALL AND JAN APELQVIST.

New procedure to treat severe leg pain

The *Silverhawk Plaque Excision System* is a new device to clean out dangerous plaque from blocked arteries in the leg. It works by using a rotating blade that shaves away plaque from the artery walls then collects it in the nosecone of the device. The plaque is then compressed so it can be removed safely from the artery. The device was approved by the FDA in 2004.

According to experts, the Silverhawk catheter removes long lesions of plaque without traumatizing blood vessels.

Ideal candidates for the Silverhawk procedure are patients with non-healing ulcers, pain in the legs when resting, simple pain when walking, gangrene and more. The procedure is minimally invasive and is performed through a tiny puncture site. ■

Chronic kidney disease and diabetes:
Amputation prevention strategies

Foot lesions in patients with diabetes mellitus and chronic kidney disease (CKD) is a problem that is frequently mismanaged—and can result in devastating consequences. Although preventable, if not treated properly and promptly, such foot lesions and related problems can lead to further complications and potentially impact a patient's survival. Improving the level of foot care, as well as properly educating patients and nephrology health care providers on proper diabetic foot care, is the first step in increasing a patient's overall survival (Broersma, 2004).

IN THE UNITED STATES, DIABETES IS THE CAUSE OF 50% OF ALL NON-TRAUMATIC LOWER EXTREMITY AMPUTATIONS AND THAT NUMBER CONTINUES TO INCREASE EACH YEAR (BROERSMA, 2004).

In fact, for the more than 40% of U.S. patients who begin chronic dialysis, diabetes mellitus is the main cause of renal failure. According to Fotieo and Reiber (1999), diabetes mellitus affected approximately 15.7 million Americans in 1999 and its complications accounted for approximately 12% of medical expenses, amounting close to $26 billion. What's more, patients with diabetes and chronic renal disease frequently present with a combination of devastating diabetes complications such as nephrology, retinopathy, and vasculopathy. Diabetic foot complications—including amputation—impact the morbidity and mortality of patients with diabetes and CKD. This is typically due to the fact that early risk factors for diabetic foot complications may be disregarded—a failure on the part of both the patient and clinician.

In the United States, diabetes is the cause of 50% of all non-traumatic lower extremity amputations and that number continues to increase each year (Broersma, 2004). Because of this, the cost of treating patients with diabetes has skyrocketed and is detrimentally affecting the patient's quality of life. Multiple factors may be responsible for the vast increase in lower extremity amputation among patients with diabetes and CKD. One reason may be related to the fact that dialysis patients often lose contact with their primary care physicians once in a dialysis setting. Therefore, many CKD patients may not receive adequate medical advice on potential foot problems.

According to Broersma (2004), preventing amputation can be achieved by having patients undergo diabetic foot examinations at least once a year to identify high-risk foot conditions. The American Diabetes Association (ADA) recommends more frequent evaluation for those patients with one or more risk factors. A visual foot inspection should take place at every doctor's visit for those patients with neuropathy, for example. Diabetic foot assessments also should be used to identify risk factors as well as other preventative measures or potential problems. According to a study by Mazze, Etzwiler, Strock, McClave, Leigh, Owens, Deebs, Peterson and Kummer (1994), **amputation rates were reduced by 28% when certain individuals were screened for high-risk foot problems and subsequently targeted with simple interventions, including patient education.**

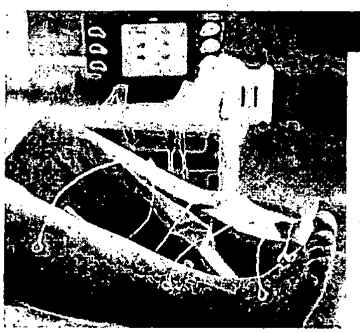
Furthermore, patients with diabetes and CKD often have frequent contact with nephrology nurses, offering them numerous opportunities for risk assessment, education, and early intervention. ■

Transcutaneous oxygen measurements
under hyperbaric oxygen conditions as a predictor for healing of problem wounds

According to Strauss, Bryant , and Hart (2002), controversy exists as to what transcutaneous oxygen ($PtcO_2$) levels are required for wound healing and what role hyperbaric oxygen has for this. Current information suggests that 30 to 40 mmHg juxta-wound oxygen tensions in room air are required.

In their paper, Strauss et al (2002) compare outcomes with $PtcO_2$ measurements in room air and with hyperbaric oxygen (HBO) in 190 patients who had wounds of the foot and ankle; then they looked retrospectively and prospectively whether there was any effect on healing. Transcutaneous oxygen measurements with HBO defined a responder group, which had a very high positive predictive value for healing of problem wounds of the foot and ankle with HBO as an adjunct to management, whether or not the wounds were hypoxic in room air.

Strauss et al (2002) conclude that $PtcO_2$ measurements under HBO have a high predictive value for healing of problem foot and ankle wounds if the readings increase to over 200 mmHg and HBO is used as an adjunct to optimal wound management. However, healing was observed in a sizable proportion of wounds that had lower readings. Consequently, juxta-wound $PtcO_2$ measurements with HBO should be used as an adjunct to the clinical evaluation. Information from $PtcO_2$ under HBO conditions predict which problem wounds will heal, whether or not adjunctive HBO is indicated, if revascularization or angioplasty is needed or should a major amputation be recommended (Strauss et al, 2002). ■



MEASURING TRANSCUTANEOUS OXYGEN LEVELS

The Radiometer TCM™400 is is a portable, noninvasive instrument that measures transcutaneous oxygen tension at up to six different points along a limb or around a wound. The TCM400 produces results that are reliable and reproducible since the instrument houses an internal barometer that automatically calculates the correct calibration value. ■

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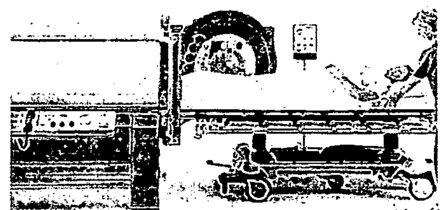
Indications for HBO



Medicare has approved reimbursement for HBO therapy when the following diagnoses are made:

- Actinomycosis
- Acute carbon monoxide intoxication
- Acute peripheral arterial insufficiency
- Acute traumatic peripheral ischemia
- Chronic refractory osteomyelitis
- Crush injuries and suture (reattachments) of severed limbs
- Cyanide poisoning
- Decompression illness
- Diabetic wounds of the lower extremities
- Gas embolism
- Gas gangrene
- Osteoradionecrosis
- Preparation and preservation of compromised skin grafts
- Progressive necrotizing infections
- Soft tissue radiation injury ■

Working with a Wound Healing Center

Wound Healing Centers are specially equipped to work with vascular patients afflicted with chronic, nonhealing wounds. Not only are the Centers uniquely equipped to administer special therapies to these vascular patients, they are also designed to help primary care physicians avoid upfront costs and time-consuming training generally associated with advanced wound care. For example, by partnering with a Wound Healing Center in offering state-of-the-art treatment modalities, your practice will:

- Provide access to clinicians with experience dealing with extensive wound population
- Ensure follow-up patient education
- Stay informed about healing progress with regular reports from our Outcomes Disease Management System ■

CONSIDER REFERRING YOUR PATIENTS TO A WOUND HEALING CENTER FOR ADVANCED WOUND CARE IF:

- A wound persists for more than 30 days with conservative treatment
- Your patient has a wound and also has circulatory problems, diabetes, or is obese
- Your patient has a wound or suffers from chronic pain and has had radiation therapy in the past
- Your patient has had a recent revascularization procedure or with a questionable vascular supply
- You are considering peripheral vascular surgical procedures or amputation

National Healing
6400 Congress Avenue
Suite 2200
Boca Raton, FL 33487

Pat Weese

From: "contact" <contact@tri-med.net>
To: "'Pat Weese'" <weese_fisher@msn.com>
Sent: Friday, June 13, 2008 10:48 AM
Subject: Emailing: Critical Limb Ischemia and Limb Salvage - The Wound Healing and Hyperbaric Center.htm

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Critical Limb Ischemia and Limb Salvage

(Exerpted from ***Wound Care Practice*, Best Publishing, Ed. Sheffield, Smith, and Fife)**
Fernando Boccalandro MD - Richard W. Smalling, MD, PhD
From the Division of Cardiology -The University of Texas Houston Medical School at Houston

Definition of Critical Limb Ischemia & Limb Salvage:

Peripheral vascular disease of the lower extremities comprise a clinical spectrum that goes from asymptomatic patients, to patients with chronic critical limb ischemia (CLI) that might result in amputation and limb loss. Critical limb ischemia is a persistent and relentless problem that severely impairs the patient functional status and quality of life, and is associated with an increased cardiovascular mortality and morbidity. It can present acutely (i.e. distal embolization, external compression, acute thrombosis, etc.) or, in the majority of cases, as chronic CLI which will be the main focus of this chapter.

Authors have proposed different definitions for chronic CLI taking into account a variety of hemodynamic measurements in combination with clinical findings, since the diagnosis based on the usual clinical manifestations of CLI (i.e. chronic non-healing wounds, resting pain, or gangrene) could also be caused by other non-vascular diseases. A practical and simple definition is the one proposed by the European Working Group on CLI. This group defined CLI as the presence of ischemic rest pain requiring analgesia for more than two weeks, or ulceration, or gangrene of the lower extremity with an ankle systolic blood pressure ≤ 50 mmHg and/or toe systolic pressure ≤ 30 mmHg.

Limb salvage can be defined as any revascularization procedure, surgical or percutaneous aimed at improving the blood flow in the ischemic limb with the purpose of preventing limb loss, and ideally achieving wound healing and resolution of chronic ischemic pain or gangrene.

Pathophysiology Of Critical Limb Ischemia

Chronic CLI in the vast majority of cases are related to advanced atherosclerotic disease. Other diseases have to be kept in mind by the clinician, specially in young patients, those with ulcers in atypical locations, or those with few or no risk factors for CLI (Table 1). Chronic CLI secondary to atherosclerosis develops when arterial stenosis reaches a critical point in which the blood flow supplied to the distal extremity is insufficient to provide the basal tissue oxygen demand. This occurs despite two compensatory mechanisms: post-stenotic arteriolar vasodilatation and development of collateral circulation. When the basal tissue oxygen demand cannot be met by the peripheral vascular system, ischemic injury occurs in the tissues with the lowest blood supply and necrosis results leading to tissue destruction, the appearance of ulceration, gangrene, and rest-pain. Besides the tissue necrosis secondary to the poor oxygen demand/supply relationship seen in these patients, they are also threatened by severe microvascular dysfunction secondary to a local and systemic inflammatory response and a thrombotic milieu that worsens their poor capillary blood flow. Also reported in this group of patients are: impaired vasomotor response, vasospasm, increased platelet aggregation, impaired fibrinolysis, abnormal healing, micro-thrombus formation, increased leukocyte activation and adhesion, increased capillary permeability with interstitial edema, local activation of the immune system with increased levels of C-reactive protein and other systemic inflammatory mediators. These seem to be accentuated in diabetics, which present with a combination of macro and micro angiopathy due to accelerated atherosclerosis, increased blood viscosity, thrombosis and an enhanced inflammatory response, which leads to a more distal and diffuse disease that might significantly limit the possibility of an effective revascularization. The presence of neuropathy in this later group also plays an important role in the pathogenesis of CLI. Neuropathy increases the risk of severe toe and foot lesions due to the absence of pain during and after trauma and the lack of early recognition of wounds that require prompt attention. It also might alter the mechanics of normal gait, worsening the perfusion on certain points of repeated pressure on the foot and toes, and predisposing some of these areas for wound formation. The lack of blood flow predisposes the ischemic tissues in diabetics to have extensive wounds with poor healing potential even after minor trauma. Diabetes mellitus predisposes the formation of early wet gangrene with polymicrobial infections that are difficult to treat due to the limited blood supply, predisposing them to the formation of deep wound infections and osteomyelitis.

Top of Page

Clinical Manifestations Of Critical Limb Ischemia

Evaluation of patients with suspected CLI is of great importance to confirm the suspected clinical diagnosis, to assess the severity of limb compromise, and to rule out the possibility of other diseases that might mimic vascular insufficiency.

The first assessment in patients with suspected CLI should always be to look for the presence of signs of acute limb ischemia that will require immediate attention and emergent revascularization. They can be remembered by the rule of the 5-"P's": Absence of Pulse, presence of resting Pain, Pallor, Paresthesia and Paralysis.

Chronic CLI is present usually in patients with previous history of intermittent claudication, smokers, diabetics, history of cerebrovascular or coronary artery disease, who now present with one or all the four hallmarks of CLI: resting pain, non healing ulcers, dry gangrene, and absence of palpable pulses.

Resting Pain: Patients with CLI usually describe their pain as a throbbing pain, dull ache, or numbness that classically worsens when the patient elevates the leg, and in the evenings or nights. It is relieved by lowering the leg to a dependent position and, interestingly, in contrast to patients with intermittent claudication, the resting pain of CLI can actually improve slightly with deambulation due to mild improvement in the arteriolar blood flow caused by the effects of gravity.

Non Healing Ulcers: Ulcers usually appear in the distal areas of the extremities such as the tips of the toes, or at bony prominences. They are associated with severe pain (except in diabetic patients with neuropathy). They are generally dry, have poor vascularity, and contain a base that can be pale, gray or black with gangrenous tissue (Figure 1).

Dry Gangrene: The presence of devitalized tissue is the end-stage clinical manifestation of CLI. It usually appears as very painful areas (except in diabetic patients with neuropathy) of necrotic and dry tissue (Figure 2). If it become infected it can present with a purulent and fetid drainage with signs of inflamation surrounding the necrotic area (wet

gangrene).

Absence Of Palpable Pulses: Palpation of distal pulses are of great importance in patients in whom CLI is suspected. Posterior tibial and dorsalis pedis pulses are almost always absent, except in selected patients with severe disease above the knee with an adequate collateral flow in whom distal pulses might be appreciated. Routinely, the physician should include examination of the popliteal and femoral pulses to localize, if possible, the area of vascular compromise. When distal pulses can not be palpated, the use of a hand-held Doppler is recommended to assess for distal blood flow.

At the bedside, measurement of an ankle systolic blood pressure using a hand-held Doppler and a regular blood pressure cuff is mandatory using the dorsalis pedis or posterior tibialis. An ankle systolic blood pressure ?50 mmHg with any of the above signs of CLI confirms the suspected diagnosis. Also a bedside ankle-brachialbrachial (ABI) index is recommended. The ABI is the ratio between the ankle systolic blood pressure and the brachial systolic blood pressure. In patients with CLI the ABI is almost universally below 0.5.

One important caveat of the ankle systolic pressure and ABI is the fact that diabetic patients with severe medial artery calcification might have falsely elevated distal pressures due to the presence of a markedly decrease in their vascular compliance. This might confuse the physician with limited experience with a discordance between the clinical presentation of the patient and the hemodynamic measurements obtained by Doppler.

In patients with palpable pulses and suspected CLI, the elevation/dependency test is a simple and accurate bedside test: the limb is elevated for 60 seconds and then lowered. In the ischemic limb the elevation and the dependency results in a purple, ruborous red color. The presence of faint pulses that disappear after a six minute walk test are also very suggestive of severe peripheral vascular disease in patients with suspected CLI and palpable pulses. The six minute walk test can be done with no equipment in the outpatient setting and its results have a good correlation with a formal treadmill test.

In an excellent review of the physical examination findings in patients with peripheral vascular disease, McGee et al. found that classic signs like abnormal resting coloration of the lower extremities, atrophic skin, lack of foot hair and abnormal capillary refill time were not associated with the presence or severity of peripheral vascular disease.

Diagnostic Test For Patients With Suspected CLI

Noninvasive vascular testing should be the next step after a thorough clinical history and physical examination. As mentioned above, an ankle systolic blood pressure and an ankle-brachial index (ABI) are two initial tests that can be easily performed at the bedside to confirm the clinical impression of CLI.
However, other tests are needed to assess the severity and anatomic localization of the sites of vascular compromise and to predict the likelihood of wound healing and need for revascularization in the case of ischemic ulcers.

Magnetic resonance arteriography (MRA): Magnetic resonance arteriography has recently become one of the preferred methods of evaluation of CLI (Figure 3). It is a non-invasive test that avoids the use of iodinated contrast and gives detailed anatomic information including graft patency and plantar archs. The use of MRA requires experienced technologists and radiologist if used as the sole pre-operative test in patients undergoing surgical revascularization. It is a technology that is evolving and continually improving.
MRA gives information not only of stenotic areas, but also provides valuable anatomic information regarding the patency of the renal arteries as well as the distal aorta, iliac and common femoral arteries that are difficult to evaluate with duplex ultrasound. Therefore the use of MRA is currently used to plan the best percutaneous or surgical strategy based not only in the stenotic segments, but moreover in the whole vascular tree of the lower extremities.

Transcutaneous Oxymetry: Tanscutaneous oxymetry measures the transcutaneous oxygen pressure at the skin surface produced by heat induced hyperemia. Different than the previous tests that give hemodynamic or anatomic information, transcutaneous oxymetry serves as a practical functional test that evaluates the oxygen delivery to the ischemic tissues. It is used not only to evaluate the need for revascularization in patients with CLI (transcutaneous oxymetry < 40 mmHg), but also predicts outcome of patients requiring amputation, survival of skin grafts, prognosis of wound healing with hyperbaric therapy and effective percutaneous or surgical revascularization.

Laser Doppler perfusion studies: As transcutaneous oxygen measurements, the use of laser Doppler perfusion with blood flow images are being used to assess tissue perfusion in compromised limbs. Its clinical use is currently limited, but has a good potential in the future for patients with CLI to determine need for revascularization, healing potential, amputation level determination, and revascularization success after percutaneous interventions.

Conventional angiography: Although it represents an invasive test, conventional contrast angiography with digital substraction is the "gold standard" for patients with CLI (Figure 4). It allows a detailed evaluation of all the different parts of the vascular tree and importantly of the distal circulation and plantar archs. It can be performed from the retrograde approach using a femoral access or antegrade through the common femoral artery or the brachial/transradial approach.

The use of digital substraction angiography that can eliminate the superimposing shadows of the underlying tissues, has enhanced the resolution of conventional angiography using modern digital technology. Emphasis must always be placed in having an adequate visualization of the distal run-offs and plantar circulation, which becomes critical in patient with CLI in which revascularization is considered. New digital angiographic systems with modern technology have improved its performance and now provide better resolution with less radiation, a better three-dimensional evaluation of the vasculature and new features that help in diagnostic and percutaneous interventions with less contrast use and radiation exposure (i.e. rotational angiography with or with out rapid computer three dimensional reconstructions, landmarking, view-trace and fluoro-trace modes, etc.).

Which diagnostic method to use? Clearly the most important is to gather a complete history and detailed physical examination with an ankle systolic blood pressure or ABI to confirm the diagnosis of CLI. Once the diagnosis of CLI is made, further tests depend largely on the experience of the center where the patient is evaluated and the clinical presentation. When resting pain or gangrene is present, a functional test is not needed and the patient should be evaluated with an MRA (or duplex ultrasound in centers with expertise in this technique) to assess the anatomic localization of the stenosis, the severity of the vascular compromise and the feasibility of revascularization. Based on this initial evaluation, a revascularization strategy is made using the preferred approach (surgical vs percutaneous) according to the patients individual situation to achieve the best short and long term results. If surgery or amputation is contemplated, MRA in experienced centers might suffice as the sole diagnostic tests. However, the information provided by the MRA is not definitive, a conventional contrast angiography with digital substraction will be needed to define the patient's vasculature and establish the best surgical technique.

For wound healing purposes, the best approach is to start with a functional test to assess the possibility of wound healing (transcutaneous oxymetry or laser Doppler perfusion) or a simple toe systolic blood pressure. If these tests show a reasonable prognosis for wound healing, a trial of intensive wound care and medical therapy should be attempted first. If this fails to achieve wound healing, or if patients have limiting intermittent claudication, or if the functional tests are suggestive of a poor healing potential; a MRA or duplex ultrasound is recommended to establish the best method of revascularization (percutaneous versus surgical revascularization). This is followed by conventional angiography if the percutaneous approach is used or if the non-invasive evaluation is inconclusive or insufficient to get the needed information before surgical revascularization.

Therapeutic Options:

Patients with true CLI have end-stage peripheral vascular disease and their therapeutic options are narrowed to either revascularization for limb salvage or amputation with its consequent limb loss. Therefore the presence of CLI is a clear indication to pursue an aggressive arterial revascularization to prevent limb loss and its associated increased mortality and morbidity. However, although revascularization represents the cornerstone in the treatment of these patients, a comprehensive team approach is needed between the primary care physician, the wound care specialist, the radiologist, the interventional cardiologist and the vascular surgeons, to offer the best care to this difficult group of patients.

The primary care physician should identify the signs and symptoms of CLI as soon as they are present, and should refer the patient as early as possible to the wound care specialist or to the vascular specialist for further assessment and aggressive treatment. The wound care specialist plays a pivotal role in the care of this patient, not only in the wound care management before and after revascularization, but also assessing the potential for wound healing and making the decision with the vascular specialists on the best timing for revascularization. All the team involved in patient care should focus in improving the patient modifiable risk factors and optimize the patient medical therapy to attempt to arrest disease progression without forgetting the high incidence of concomitant heart and cerebrovascular diseases seen in this group of patients. The vascular specialist (interventional cardiologist/radiologist or vascular surgeon) must make the best decision regarding the preferred revascularization approach based upon the disease segment to treat, the inflow and outflow in the affected limb and the underlying operative risk to the patient. Three forms of revascularization therapy are available for patients for limb salvage: surgical revascularization, endovascular therapy and thrombolysis.

Surgical Revascularization:

Currently there are different surgical techniques that allow long-lasting lower extremity revascularization for patients suffering from CLI. The surgical treatment depends on the anatomical area to revascularize (aorto-iliac, femoral or below the knee), the type of conduit used (venous versus prosthetic) and quality of the graft inflow and outflow runoffs. In different surgical series with unselected patients, limb salvage has been reported to be between 65 to 80%. Better success is attained when the lesion is higher in the vascular tree, with best outcomes in aortoiliac disease and worst outcomes when disease is present below the knee.

The type of graft in CLI is also an important consideration, prosthetic grafts are successfully used for aorto iliac disease with patency at 5-years close to 90%. However, the use of venous grafts have significantly improved patency rate compared with prosthetic grafts when anastomosed at the knee level (68% versus 38% at 5 yr.) or below (50% versus 12% at 5 yr); and therefore should be the preferred conduit for these anatomic sites.

One of the most important aspects of graft patency depends on the distal run-off. Patients with severe distal disease, poor pedal arches, and slow or poor distal run-offs have a very low patency rate. Thus, it is generally recommended to have at least two vessel runoffs before surgical revascularization is considered. Besides the quality of the runoffs and the severity of the vascular disease, graft failure is not uncommon in patients undergoing limb salvage due to spontaneous thrombus formation, disease progression and graft or anastomotic neointimal hyperplasia. To limit disease progression, an aggressive medical therapy should be instituted to try to the control risk factors and improve lipids, glucose, smoking and blood pressure control. The use of pharmacologic therapy to prevent graft failure remains uncertain, except for the use of antiplatelet agents that are recommended (i.e. aspirin and clopidrogel) and, in some cases, of prosthetic grafts going to the femoropopliteal or below. The use of coumadin is also advocated.

Top of Page

Endovascular Therapy:

In the past two decades, endovascular therapy has revolutionized the treatment of patients with vascular disease. In this group of patients with multiple medical problems, advanced age and high surgical risk, endovascular therapy is playing a leading role in providing effective revascularization and limb salvage, while limiting the operative risks as compared with vascular surgery.

Although advances have been made in innovative technologies with emphasis in lesion modification, atheroablation and other ingenious approaches, the mainstay of endovascular therapy still is balloon dilatation and stenting (Figure 4).

Outcomes of endovascular therapy as in surgical revascularization are dependent on the distal run-off. mproved outcomes are also seen in the iliac vessels as compared with femoro-popliteal or distal disease revascularization procedures. Higher success rates are seen in patients with focal and short stenosis, with mild diffuse distal disease, in non-diabetics, in patients with reconstituted pedal arches after the procedure, and in non smokers.

The 5-year patency rates for aortoiliac disease have been reported close to 85% with stent implantation and for femoropopliteal disease, between 38 to 70% in combined series of balloon angioplasty with and with-out stenting. Although the immediate angiographic and clinical success are usually good, the long-term patency of these procedures are jeopardized by restenosis, especially at the femoropopliteal level and below the knee, requiring in many cases a repeat revascularization procedure to reestablish and maintain vessel patency.

For infrapopliteal disease, the use of angioplasty with coronary techniques have demonstrated generally a poor durability, but nevertheless its recognized role in limb salvage is now accepted (up to 80% of limb salvage was produced by Bakal et al. in carefully selected patients), preventing limb amputation and improving wound healing even in patients that are not candidates for surgical revascularization.

There have been no randomized studies comparing the current endovascular therapies with surgical revascularization for CLI, but data from different comparative studies and from series of patients suggest that they might have a similar outcome regarding limb salvage. However, the patients undergoing endovascular therapy might require repeat revascularization procedures due to the presence of restenosis to maintain patency, with the advantage of avoiding an initial surgical procedure. In patients undergoing endovascular therapy for CLI, it is also very important to aggressively modify their risk factors and maintain an adequate antiplatelet regimen with aspirin and most likely also combined with clopidrogel for long term.

Amputation:

For patients who are not candidates for revascularization, amputation is an important treatment option. The level of amputation and the potential for effective rehabilitation are the two main factors to take into account. More distal amputation has a better rehabilitation potential, but also have a higher risk of incomplete healing with a sub-optimal treatment, resulting in further amputations. Therefore, before the level of amputation is chosen it is important to consider the following factors besides the overall cardiopulmonary condition of the patient: Non-invasive or invasive assessment of the arterial perfusion in the affected limb to guarantee effective healing (usually a calf pressure > 70 mmHg or an ankle pressure > 30 mmHg are considered adequate for healing after amputation), the presence or absence of infection (i.e. celullitis, osteomyelitis, etc), glucose control in diabetics, adequate nutrition, and attention to any mechanical feature that might compromise wound healing after the amputation.

With the current techniques must patients with CLI can benefit from revascularization. Initially, the percutaneous approach is preferred by many centers in order to avoid a surgical procedure in a group of patients that are at high operative risk. However, the best treatment to achieve the highest rates of limb salvage and long-lasting revascularization can not be generalized; and in the best interest of the patient, needs to be individualized based on their underlying risk factors, comorbidities, severity of peripheral vascular disease and particular anatomy.

Top of Page

Future Treatment Options for Limb Salvage

Many efforts are devoted to try to improve the revascularization outcomes in patients suffering from CLI. Use of debulking techniques with rotational atherectomy has been disappointing. Effectiveness of laser angioplasty for limb salvage is still undetermined, although it also has been disappointing in other vascular applications. One area of intense research is the prevention of restenosis in the peripheral vasculature in which the use of drug eluting stents holds a bright future. Other emerging strategies to prevent or treat restenosis include the use of beta or gamma radiation, platelet derived growth factors (PDGF), angiopeptin, arterial gene therapy with nitric oxide donors, stent gene delivery, drug electroencapsulation, and crio or photoangioplasty.

With this endovascular armamentarium, also novel pharmacologial approaches are being studied to try to improve the restenosis rates (i.e: iloprost and prostaglandin derivates, cilostazol, low molecular weight heparins and direct thrombin inhibitors, etc) and improved vascular growth and collateral vessels formation (gene therapy and vascular growth factors). Also newer graft materials are being tested and some novel techniques in which gene delivery is combined with new prosthetic graft materials are being developed to prevent graft thrombosis and to improve graft patency.

Protocol For Limb Salvage

As a guide for the clinician, we provide this simple algorithm to help in the management of patients with CLI:

PROTOCOL FOR CHRONIC CRITICAL LIMB ISCHEMIA

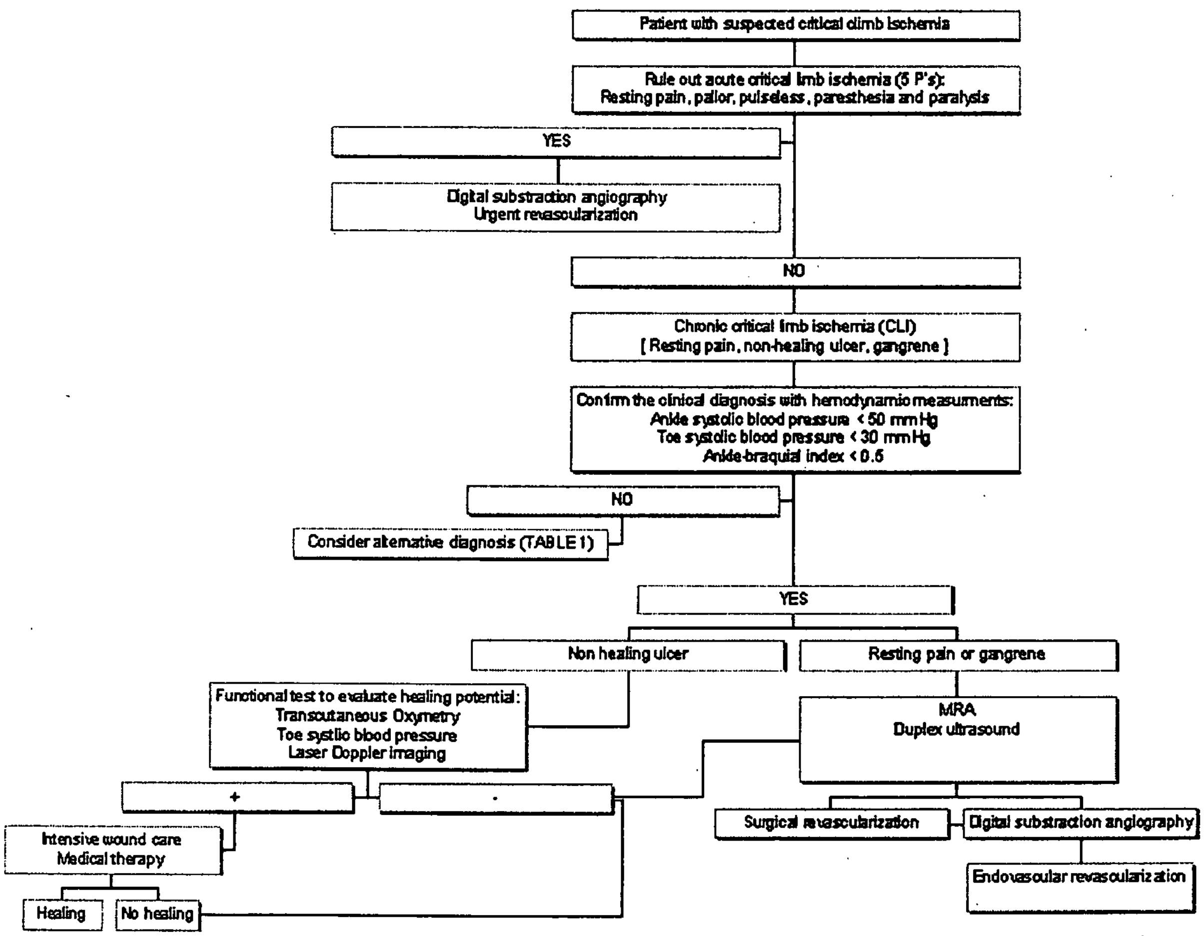


Table 1: Differential diagnosis for chronic critical limb ischemia.
Atheroembolism.
Cardioembolism.
- Atrial fibrillation.
- Patent foramen ovale.
- Left ventricular thrombus.
- Endocarditis.

Drugs induced.
Ergotamine abuse.
Hypercoagulable states.
Homocystinemia
Antithrombin III deficiency
Protein C and S deficiency
Insect bites
Brown recluse spider.
Parasitic diseases
Filariasis.
Radiculopathies and spinal stenosis.
Systemic vasculitis.
Polyarteritis nodosum
Thromboangitis obliterans
Wegner's disease
Essential cryoglobulinemia
Takayasu's disease
Giant cell arteridities
Sympathetic dystrophy
Vasospastic disorders
Raynaud's phenomenon

Top of Page

Images

FIGURE 1:
Non-healing ulcer in a patient with chronic limb ischemia.

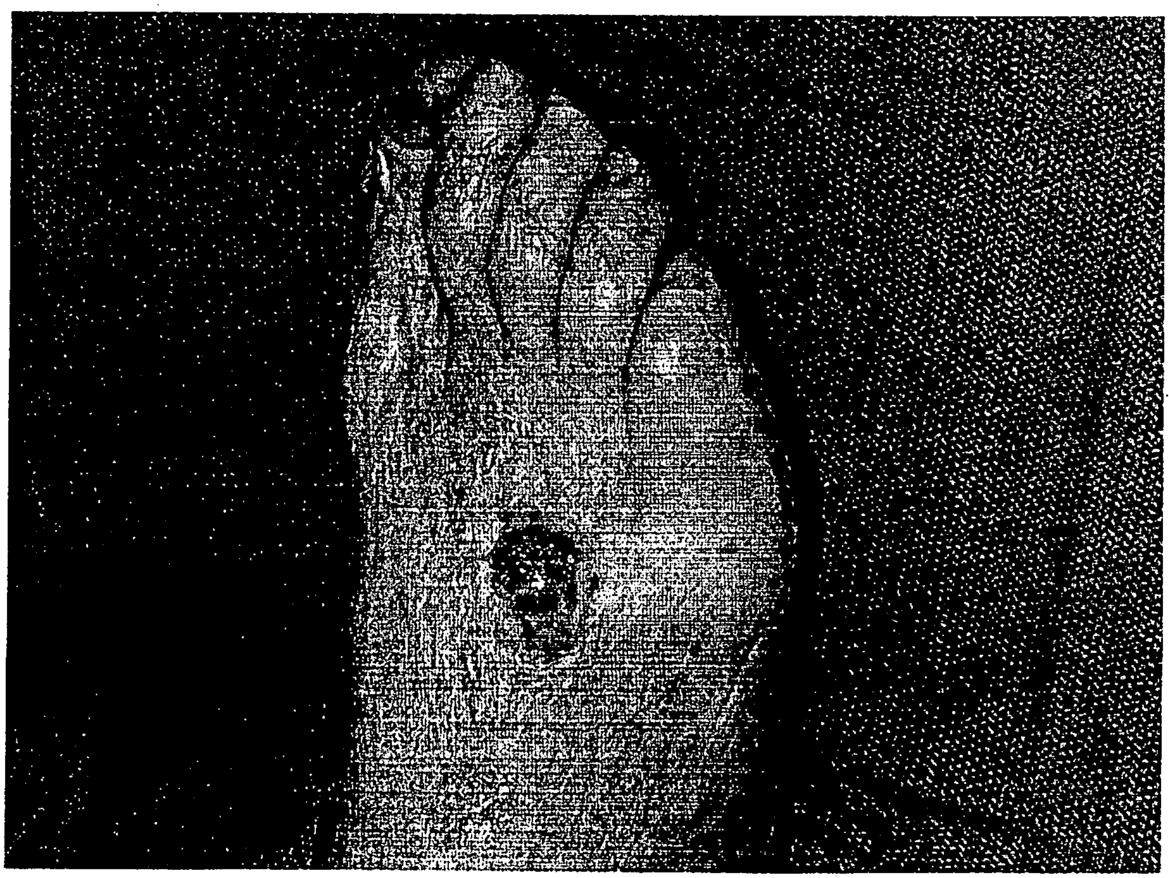


FIGURE 2:
Gangrenous foot due to severe advanced chronic limb ischemia in a diabetic patient.

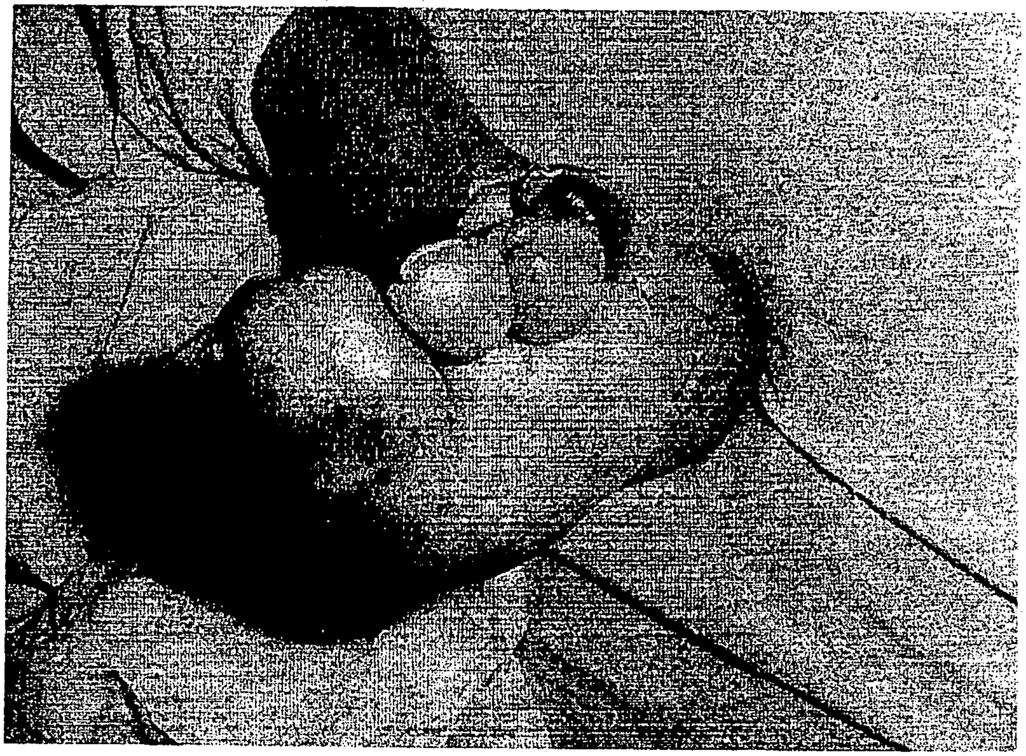


FIGURE 3:
Magnetic resonance angiography showing bilateral total superficial femoral occlusions in a patient with a non-healing ulcer of the right lower extremity. (CFA = Common femoral artery, PFA = Profunda femoral artery, SFA = Distal superficial femoral artery filling from collateral flow from the profunda femoral artery with no visible proximal run-off).

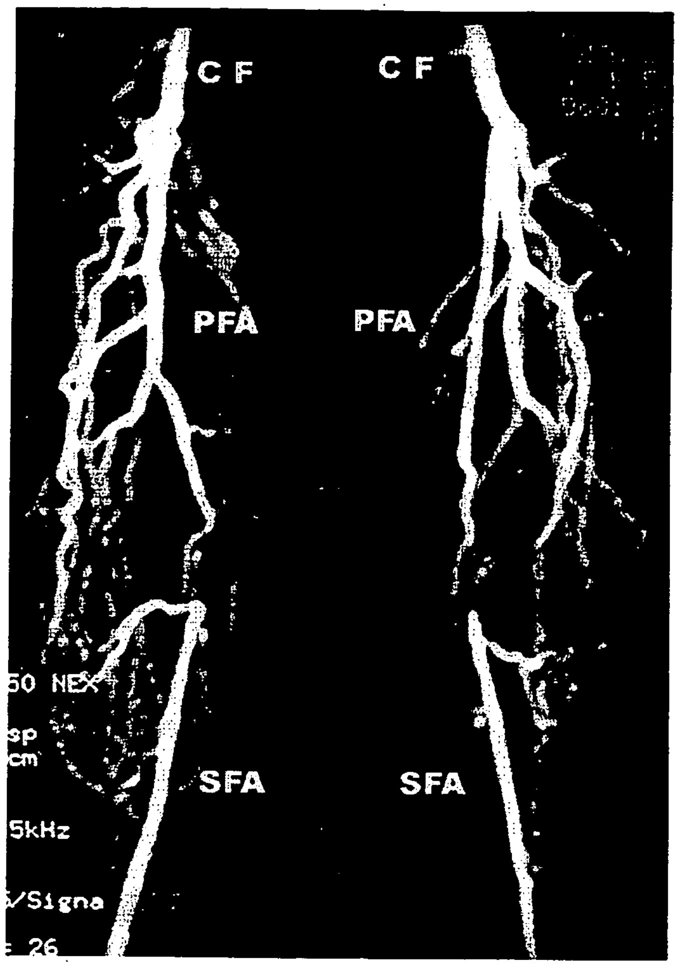


FIGURE 4:
(a) Digital substraction angiography of the right leg of the patient in Figure 3 showing a stump in the take off of the superficial femoral artery.

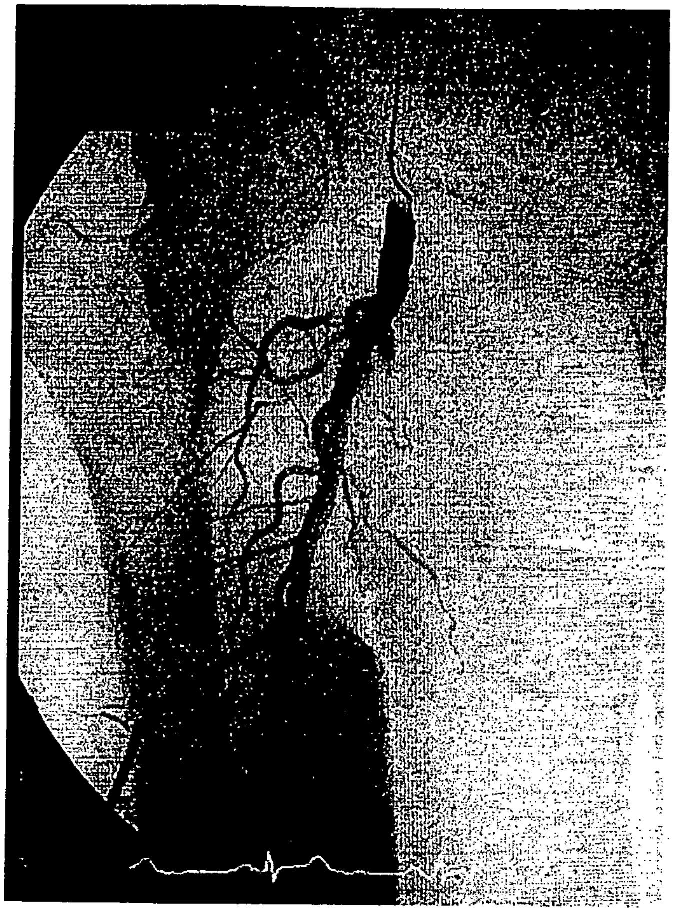
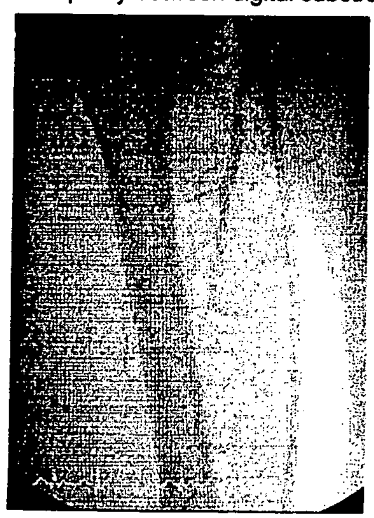


FIGURE 4:
(b) Regular angiography in the same patient following a percutaneous retrograde superficial femoral artery reconstruction using the popliteal approach. Note the nitinol stents placed in the superficial femoral artery with its reconstituted runoff and the differences in the quality between digital substracted and regular angiography.



Top of Page

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Top of Page

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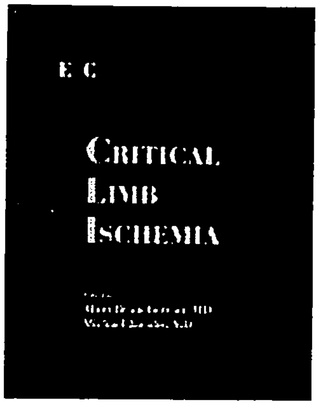

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This book contains 28 chapters representing the opinion of key vascular specialists. It covers a wide spectrum of definition and criteria of critical limb ischemia (CLI), its pathophysiology, epidemiology, assessment, and therapy. Objective criteria, which are associated with the definition of CLI, have been the source of many debates and, therefore, their disadvantages and limitations are presented and alternatives and recommendations are suggested.Present-day medical treatment of CLI includes spinal cord stimulation, antiplatelet drugs, defibrinating agents, vasoactive substances, and prostanoids. Therapies of choice, such as distal bypass surgery for extensive arterial disease, and alternative procedures are also surveyed.Clinical and surgical specialists concerned with endovascular and surgical intervention will benefit from this engaging treatise that stretches from general to specific medical conditions of CLI, quality of life, long-term morbidity and mortality, and the role of primary amputation.

Contents

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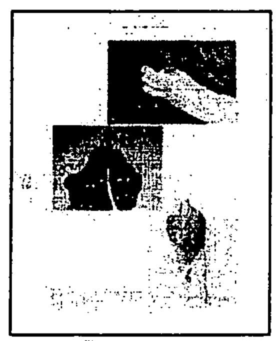
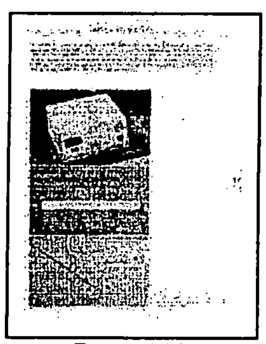
Page 231



Page 232



Page 113

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